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THE USE OF ANTICOAGULANTS IN THE TREATMENT AND PREVENTION OF CORONARY ARTERY DISEASE AND MYOCARDIAL INFARCTION*

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RATIONALE

The use of dicumarol and heparin in acute coronary thrombosis and myocardial infarction, in order to reduce both mortality and morbidity, is gaining favor. Recent carefully conducted studies by Blumgart and co-workers[1], and LeRoy and Nalefski[2] successfully demonstrate that the use of dicumarol in dogs in which coronary occlusion has been produced does not increase the tendency to hemorrhage in the infarcted myocardium, neither does it alter the size of the infarct, nor produce any other deleterious effect on the myocardium. In both treated dogs and controls no mural thrombi were found. Beattie and co-workers[3] had previously conducted similar experiments in dogs and found no differences in the gross or histological pathologic findings in the dicumarolized group compared with the control animals. They stated hemorrhage was not a prominent feature in either group of animals, and concluded there was no evidence that dicumarol had a favorable effect on experimental myocardial infarction. These studies, conducted by independent investigating groups serve to allay the fear of rendering the infarct excessively hemorrhagic or extending its size when anticoagulants are employed in patients with acute myocardial infarction.

The following objectives obtain in using anticoagulants in acute myocardial infarction:

1. Prevention of propagation of the initial coronary thrombosis with subsequent increase in the size of the infarct.

2. Prevention of a complicating new coronary thrombosis and/or myocardial infarction during the healing stage of the initial attack.

3. Prevention of intracardiac mural thrombi, with subsequent peripheral embolization from the left side of the heart, to the brain, kidney, spleen, mesentery, extremities, or even a coronary branch, and avoiding those pulmonary emboli which derive from the right cardiac chambers.

4. Prevention of phlebothrombosis in the leg veins, the prime source of subsequent pulmonary emboli.

5. Prevention of concomitant arterial thrombosis in the brain, lungs, abdominal organs, and extremities.

During the acute attack, the enforced bed rest and fall in blood pressure contribute to the likelihood of thrombo-embolization. If congestive heart failure ensues, thromboembolic lesions are fostered by the pathophysiology as well as by the usual treatment for heart failure.

Bean[4] found in 300 autopsied cases of myocardial infarction that embolism was a significant factor in causing death in 26 per cent. In 100 cases of acute myocardial infarction studied by Nay and Barnes[5] vascular complications were found in 37 per

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cent. Hellerstein and Martin[6] report 45 per cent incidence of thrombo-embolism in 160 autopsied cases of myocardial infarction. Ogura and associates[7] found peripheral thrombo-embolic phenomena in 45 per cent of 100 cases of myocardial infarction at autopsy, with proximal extension of the coronary thrombosis in 12 per cent and additional coronary thrombosis in 19 per cent of the cases. Mintz and Katz[8] reported that 9.9 per cent of 572 patients with acute myocardial infarction had clinical thrombo-embolic lesions. Foord[9] found mural thrombi in 40 per cent and embolic lesions in 25 per cent of 442 cases of autopsy in which there was old or recent cardiac infarction, and he mentions unpublished data of Edmondson revealing 328 thrombo-embolic manifestations in 1,000 cases of acute myocardial infarction. A compilation of reports on 924 cases of myocardial infarction by Hellerstein and Martin[6] augmented by the data of Ogura[7] and Foord[9], reveals mural (intracardiac) thrombi were manifest at autopsy in nearly 45 per cent of 1466 cases. Recently Adams and Cohen[10] found myocardial infarction to be the third commonest cause of cerebral embolism in a study of 120 cases of cerebrovascular lesions at autopsy.

Bean[11] found that myocardial infarction developed in 20 per cent of cases as a consequence of organic narrowing of the coronary artery without thrombosis. Commenting on this, and the fact that coronary thrombosis may be initiated by intimal hemorrhage in a coronary artery, he states: "Where these mechanisms prevail a reduction of coagulability of the blood would be ineffective and might even be dangerous." The fear that anticoagulants might induce coronary artery intimal hemorrhage and thus aggravate or initiate coronary thrombosis has been quieted by the failure to discover any significant degree of coronary subintimal hemorrhage at autopsy in cases treated with anticoagulants, both in the study of 90 autopsied cases by the American Heart Association Committee, and in 20 autopsies in my own acute infarction series and long-term "coronary" cases, in whom careful histologic studies of the myocardium and coronary vessels were made by Dr. Philip Rezek.* As for the use of anticoagulants in those myocardial infarcts not caused by coronary thrombosis, such an infarct is just as likely to be complicated by mural thrombosis and subsequent peripheral thrombo-embolism as is the case with myocardial infarction caused by coronary occlusion.

To ward off propagation of the coronary thrombus and to prevent secondary thrombosis or infarction during the lag period of dicumarol, heparin is given the first few days and to prevent mural thrombi as well, although pathologic studies indicate that mural thrombi seldom form until the fourth day after myocardial infarction. Heparin in retarding media may be used throughout the four to six weeks of the acute episode, as advocated by Loewe[12] without recourse to dicumarol.

RESULTS OF TREATMENT OF ACUTE MYOCARDIAL INFARCTION

The efficacy of the treatment of acute coronary thrombosis and/or acute myocardial infarction with anticoagulants has descended from the realm of speculation to the firm ground of statistical proof following the laborious and splendid compilation by Wright, Marple and Beck[13] of the results of the cooperative study by the American Heart Association Committee for the Evaluation of Anticoagulants in the Treatment of Coronary Thrombosis with Myocardial Infarction. The final report of this committee has not been published but the augmented data in 589 "treated" cases compared to a "control" group of 442 cases reveals the mortality rate in the treated group was 16 per cent compared to 23.4 per cent in the control group; while in the treated group 10.9 per cent developed thrombo-embolic complications compared to 26 per cent in the control group.

Since June 1943, up to May, 1949, I have used anticoagulants in 160 patients in 166 attacks of acute myocardial infarction, excluding ward cases incorporated in the American Heart Association Committee

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study. The results in 68 attacks have been reported,[14] while the mean death rate in all attacks was 13.2 per cent (Table I), the mortality rate in 99 first attacks was 6 per cent and in patients with one or more previous attacks was 25.3 per cent. Furman[15] and co-workers recently stated that in 82 patients with myocardial infarction receiving anticoagulants the reduction in mortality was primarily in first attacks.

TABLE I

AUTHOR'S CASES OF ACUTE CORONARY THROMBOSIS TREATED WITH ANTICOAGULANTS, JUNE 1943-MAY 1949

	Number		Deaths
1st Attacks	99		6
2nd "	59		15
3rd "	7		1
4th "	1		1
160 Patients	166 Attacks	23	(13.8%)
Autopsies	14	Thrombo-embolism	4
Mural thrombi	2	Subepicardial hemorrhage	3
Major hemorrhage	16	Cardiac rupture	1

Only 4 patients developed thrombo-embolic complications while taking anticoagulants. In one case there was inconclusive clinical evidence of pulmonary embolism, and in another although autopsy disclosed mesenteric embolism, this apparently derived from an aortic atheromatous plaque as no intracardiac thrombi were found. A third case with extensive infarction dying with congestive failure after four weeks had a small mural thrombus and bilateral renal emboli at autopsy. A fourth case with old and recent myocardial infarction was started on heparin which was discontinued because of hematemesis. Two weeks later pulmonary embolization developed so heparin was used again without further bleeding. Ventricular tachycardia developed followed by femoral embolism and then cerebral embolism, both clearing clinically when heparin was augmented. Death later ensued from congestive failure and autopsy showed extensive cardiac infarction with mural thrombosis.

Microscopic sections of the myocardium in 14 autopsied cases did not reveal any significant adverse effects attributable to use of anticoagulants, except for hemorrhage between the media and adventitia in one coronary artery branch, mild subendocardial hemorrhage in 3 cases, 1 of which also showed hemorrhage within the infarcted myocardium. No toxic effects were found in the liver and kidneys.

Major hemorrhagic complications, chiefly gross hematuria, developed in 10 per cent of this series. There were no deaths definitely due to hemorrhage, although 1 case not autopsied may have had a cerebrovascular accident, and 1 patient who developed ventricular rupture of course had hemopericardium.

TABLE II

MORTALITY IN ACUTE CORONARY THROMBOSIS AND MYOCARDIAL INFARCTION TREATED WITH ANTICOAGULANTS

JUNE 1943-MAY 1949

Author	Number of Cases	Deaths
Nichol	166*	23
Peters et al	110	12
Wright	76	15
Parker-Barker	100	11
Fuller	34	5
Reich-Eisenmenger	24	4
McCall	71	9
Greisman-Marcus	75	7
Total	656	86 or 13.1%

Reports exclusive of Am. Heart Ass'n. Study so data of Glueck et al, Van der Veer-Marshall, omitted.

*98 added to previous reports of author.

Table II summarizes the results in 656 "treated" cases reported to date *not* incorporated in the cooperative Committee study. The mortality rate averaged 13.1 per cent; whereas in 2,325 cases of acute myocardial infarction, not given anticoagulants, recorded in the literature since 1940 the mean mortality was 28.8 per cent (Table III). Table IV shows the low incidence of thrombo-embolic complications, mural thrombi, and ventricular rupture in the treated groups.

DISCUSSION

It is with astonishment one hears Murray[16] recently remark, anent acute myocardial infarction, "medical treatment is so ineffective, and is entirely helpless, except from a palliative point of view" especially as Murray pioneered in the use of heparin

TABLE III

IMMEDIATE MORTALITY MYOCARDIAL INFARCTION REPORTS SINCE 1940

Author	Cases	Death %
Rosenbaum-Levine	208	33
Bland-White	86	44
Rathe	274	20
Smith et al	100	15
Woods-Barnes	128	46
Shillito et al	50	34
Nay-Barnes	100	13
Baer-Frankel	378	39
Peters et al	86	25
Fisher-Zukerman	108	33
Chambers	100	34
Fuller	35	28
Mintz-Katz	572	21
Greisman-Marcus	100	35
Total	2,325	28.8%

in surgical cases.

Parker and Barker[17] recently commented: "Intravenous injections and daily venepunctures are not to be considered lightly in the management of patients with acute myocardial infarction, when complete rest, freedom from apprehension and the avoidance of unessential therapeutic and diagnostic procedures have long been accepted as imperative. To justify its use, one must certainly show sufficient value in any diagnostic or therapeutic procedure which is mentally or physically disturbing to the patient."

Since increase in hemorrhage and softening of the myocardial infarcted area with consequent rupture was one of the dire consequences commonly predicted would follow the use of anticoagulant therapy, it is of no little interest that in the American Heart Association study only a few cases had myocardial rupture, and it will be seen in Table IV that only 5 cases were discovered at autopsy in 580 treated cases. Thus the incidence of cardiac rupture was much lower than the 4 to 6 per cent rate commonly reported. It might be postulated that there is less abrupt infarction, with improved collateral blood flow, and less necrosis in the infarcted area resulting in less loss of tensile strength of the myocardium, all of which factors enter into the pathogenesis of cardiac rupture.

Comparison of the death rate in untreated patients and those receiving anticoagulants, though statistically highly significant, does not reflect the true value of anticoagulant therapy as much as the reduction of thrombo-embolic lesions with subsequent morbidity.

TABLE IV

INCIDENCE OF THROMBO-EMBOLIC COMPLICATIONS IN 580 CASES CORONARY THROMBOSIS TREATED WITH ANTICOAGULANTS

Author	Cases	Thrombo-Embolism	Autopsy	Mural Thrombi	Cardiac Rupture
Nichol	166	4	14	2	1
Peters et al	110	3	5	1	1
McCall	71	2	3	0	1
Parker-Barker	100	5	7	2	2
Fuller	34	2	0	?	?
Reich-Eisenmenger	24	1	0	?	?
Greisman-Marcus	75	3	1	0	0
Total*	580	20	30	5	5

*Wright's 76 cases not included as he *chose* cases with T. E.

It is now possible to confidently attend patients with acute myocardial infarction, secure in the knowledge that proper heparin-dicumarol therapy will greatly minimize one prime failure of the usual treatment i. e., the development of thrombo-embolic complications, the full import of which will especially impress cardiologists inured to the tragic development of a fresh myocardial infarction or cerebral, pulmonary, or peripheral thrombo-embolization, just when recovery from the primary insult seemed assured.

LONG-TERM PROPHYLAXIS

As coronary thrombosis in dogs was shown by Solandt and Best[18] to be prevented by the use of heparin, it is conceivable that coronary thrombosis may be prevented in clinical practice by the continuous use of dicumarol.

The premonitory signs of an acute attack are often absent and when present are frequently misinterpreted except in retrospect, and as detection of the crucial point when anticoagulants are required as a preventive measure is often impossible, dicumarol must be used continuously after recovery from an acute attack if an attempt to forestall *recurrent* coronary thrombosis is to be undertaken.

After the first month of dicumarol therapy during the acute episode the time interval elasping between prothrombin tests was extended to twice a week, and by the end of the second month was often extended to once a week. After six months in some patients the tests were made at ten day intervals rarely at fifteen day intervals in patients exceptionally well "stabilized." Occasionally unexplained variations occur in tolerance for dicumarol even after two or three years treatment, so it is *never* advisable to extend the testing interval beyond fifteen days. Only reasonably intelligent patients were selected and all were well instructed in the purpose of treatment with dicumarol and the risk if directions were not followed. Urinalysis was done once or twice a month to detect hematuria, and liver function studies were made approximately every six months in most cases.

A preliminary report[19] in 1946 described the first cases managed in this fashion. Foley and Wright[20] have lately reported success with similar long-term therapy. Thirty-four patients have followed the regime for three to twelve months and 20 patients from one year to five years (54 cases in all). Five patients discontinued therapy after three to twenty-three months. Nine patients died while under treatment. Autopsy was performed in 6 cases, revealing fresh coronary thrombosis or infarction in 2 cases and cerebral hemorrhage in 2 others. In 5 cases death was ascribed to either ventricular fibrillation, acute coronary insufficiency or congestive heart failure. No mural thrombi or other complicating thrombo-embolic manifestations were found.

Major hemorrhage developed in 10 cases. Patients taking dicumarol for months or years run more risk of hemorrhage than patients taking dicumarol a few weeks only, and 2 developed fatal hemorrhage. One patient died with cerebral hemorrhage which probably was not induced by dicumarol as the right lenticulostriate artery was ruptured, and it is unlikely that he would have survived the eight moths he did after 3 attacks of myocardial infarction without dicumarol in view of his poor condition. Another hypertensive patient with marked cerebral arteriosclerosis and congestive heart failure died in convulsions and coma after two months of dicumarol therapy and at autopsy petechial hemorrhages were found in the capsule and pons.

SUMMARY

The rationale and the results of anticoagulant therapy in acute myocardial infarction have been reviewed. Data on 656 "treated" cases reported to date, including 98 additional cases of the author, none of which were incorporated in the American Heart Association Committee Study are presented. The mortality rate was 13.1 per cent and exceedingly few thrombo-embolic complications were encountered. These data compared favorably with the American Heart Association Committee Study, but lack a definite control group, although analysis of 2,325 cases reported since 1940 not receiving anticoagulants revealed a mortality of 28.8 per cent.

Long-term dicumarol therapy in 54 cases of the author, treated from three to sixty-one months in order to prevent recurrent attacks of myocardial infarction, is reported briefly and although no control series existed the results encourage further trial of long-term prophylaxis.

REFERENCES

1. Blumgart, H. L., Freedberg, A. S., Zoll, P. M., Lewis H. D. and Wessler, S.: The effect of dicumarol on the heart in experimental acute coronary occlusion. Tr. A. Am. Phys. 60:228, 1947.

2. LeRoy, G. V. and Nalefski, L. A.: Dicumarol in experimental myocardial infarction. J. Lab. & Clin. Med. 33:961, 1948.

3. Beattie, E. J., Cutler, E. C., Fautex, M., Kinnery, T. D. and Levine, H. D.: The use of dicumarol in experimental coronary occlusion. Am. Heart J. 35:94, 1948.

4. Bean, W. B.: Infarction of heart: Clinical course and morphological findings. Ann. Int. Med. 12:71, 1938.

5. Nay, R. M. and Barnes, A. R.: Incidence of embolic or thrombolic processes during immediate convalescence from acute myocardial infarction. Am. Heart J. 30:65, 1945.

6. Hellerstein, H. K. and Martin, J. W.: Incidence of thrombo-embolic lesions accompanying myocardial infarction. Am. Heart J. 33:443, 1947.

7. Ogura, J. H., Fetter, N. R., Blankenhorn, M. A. and Glueck, H. I.: Changes in blood coagulation following coronary thrombosis measured by heparin retarded clotting test (Waugh and Ruddick test). J. Clin. Investigation 25:586, 1946.

8. Mintz, S. and Katz, L. N.: Recent myocardial infarction. Arch. Int. Med. 80:205, 1947.

9. Foord, A. G.: Embolism and thrombosis in coronary heart disease. J. A. M. A. 138:1009, 1948.

10. Adams, R. D. and Cohen, M. E.: Vascular diseases of the brain. Bull. New England M. Center 9:184, 1947.

11. Bean, W. B.: Anticoagulants in the treatment of acute myocardial infarction and its complications. J. Iowa S. M. Soc. 39:149, 1949.

12. Loewe, L.: Anticoagulation therapy with heparin Pitkin menstruum in thrombo-embolic disease. Am. J. Med. 3:447, 1947.

13. Wright, I. S., Marple, C. D. and Beck, D. F.: Report of the committee for the evaluation of anticoagulants in the treatment of coronary thrombosis with myocardial infarction. Am. Heart J. 36:801, 1948.

14. (a) Nichol, E. S. and Page, S. W., Jr.: Dicumarol therapy in acute coronary thrombosis: Results in fifty attacks. J. Florida M. A. 32:365, 1946.

(b) Nichol, E. S.: Treatment of acute coronary thrombosis with dicumarol: Further observations. Summarized in Am. Heart J. 33:722, 1947.

15. Furman, R. H., Gale, R. G., Billings, F. T., Jr., and Meneely, G. R.: Critical appraisal of anticoagulant therapy with heparin and discumarol in coronary arteriosclerosis with myocardial infarction by comparison within prognostic categories. Abstracted in Am. Fed. Clin. Research, July, 1949.

16. Murray, G.: The pathophysiology of the cause of death from coronary thrombosis. Ann. Surg. 126:523, 1947.

17. Parker, R. L. and Barker, N. W.: The effect of anticoagulants on the incidence of thrombo-embolic complications in acute myocardial infarction. Proc. Staff Meet., Mayo Clinic 23:367, 1948.

18. Solandt, D. Y. and Best, C. H.: Heparin and coronary thrombosis in experimental animals. Lancet 2:130, 1938.

19. Nichol, E. S. and Fassett, D. W.: An attempt to forestall acute coronary thrombosis: Preliminary note on the continuous use of dicumarol. South. M. J. 40:631, 1947.

20. Foley, W. T. and Wright, I. S.: Long term anticoagulant therapy for cardiovascular disease. Am. J. M. Sc. 217:136, 1949.

VAGOTOMY IN THE TREATMENT OF PEPTIC ULCERATION

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NEW ORLEANS

Vagus nerve resection for peptic ulcer has not gained professional enthusiasm in the city of New Orleans. This is reflected in the fact that just 4 such operations have been performed at Charity Hospital, 6 at Ochsner Clinic, 4 at Touro Infirmary, 13 at Baptist Hospital, 1 at Lakeshore, and none at Hotel Dieu. Our own experience* with the procedure is limited to 4 vagus nerve resections performed transthoracically and 2 performed subdiaphragmatically, without combination with any other operative procedure.

I would like to report our results along with those of others in the city, to compare them with the results published in the literature, and to discuss the controversial issue as to what place vagus nerve resection has in the surgical treatment of peptic ulcer.

Animal experimentation was begun nearly sixty years ago, and thirty-seven years have elapsed since Exner performed his bilateral vagotomies in man. It was soon learned that the incomplete operation accomplishes little, while the complete operation causes gastric hypomotility, a complete absence or a marked diminution in the free hydrochloric acid, and a great reduction in the amount of resting gastric secretion. Clinically, the operation usually produces a dramatic relief from ulcer pain, prompt healing of the ulcer, but undesirable side effects of disturbed motility in a significant percentage of cases.

RESULTS

The results in our 6 cases were satisfactory. All were cases of duodenal ulcer with complications which indicated surgical intervention. The postoperative morbidity was low with the exception of 1 case of atelectasis which required bronchoscopic aspiration. In each case ulcer pain was relieved. X-rays taken postoperatively showed healing of the ulcer. The insulin tolerance test was negative in 5 cases and was equivocal in the sixth inasmuch as the hypoglycemia was not adequate. The postoperative acid levels varied from a complete absence of free hydrochloric acid to only a moderate hypoacidity. One patient threatened to have major symptoms of gastric retention which disappeared with an adjustment in diet. A second patient exhibited transient symptoms of esophageal spasm. Both of these patients were rather pleased that

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their chronic constipation appeared to be abolished by the operative procedure, but there was no troublesome diarrhea in any case. The postoperative sequelae were so mild in comparison with the preoperative ulcer symptoms that every patient was quite satisfied. After an average follow-up period of approximately nine months, there has been no significant change except 1 patient who now, after fourteen months, has regained his gastric motility, and his formerly complete anacidity is now four clinical units, although he remains asymptomatic and these changes have occurred in spite of a negative insulin tolerance test. This is in keeping with the observation of Moore who found that a year after vagectomy, in some cases the stomach partly recovered its motility and acidity in the presence of repeatedly negative insulin tolerance tests, which led him to conclude that this represented autonomy of the stomach, and that the beneficial effects which still persisted did so because the cephalic phase of secretion was interrupted, and stress and nervousness could no longer be transmitted to the stomach.

Of the 4 Charity Hospital cases, 2 were unsatisfactory. One of these exhibited the highest degree of gastric stasis in the series, amounting to as much as 1500 cc. of fluid, and required the daily passage of a Levine tube for about nine months. This gastric stasis occurred in spite of a pyloroplasty performed concomitantly with the vagotomy because of a recognized pyloric obstruction. Of the 6 Ochsner Clinic cases, only 4 consisted of the nerve resection alone and, of these, Penick considers only 1 to be satisfactory after a year's observation. Mahorner has performed the majority of the vagotomies in New Orleans but he has used it, as has Crile at the Cleveland Clinic, as a procedure to complement gastric resection in many of his cases and considers his results satisfactory in nearly every patient. O'Neil's several vagotomies consisted of about an equal number of satisfactory and unsatisfactory results. However, the latter seemed to be mainly psychoneurotic symptoms when postoperative x-rays showed no presence of an ulcer. In summary, from a study of the records and from personal communication with the surgeons who were acquainted with the majority of the cases performed in this city, there was not enough information to attempt an accurate statistical grading. However, it appears that less than 75 percent of the cases were completely satisfactory. There was no mortality for vagus resection alone, nor was there an instance of persisting ulcer or of recurrent ulceration in the patients whose records were available.

The results obtained by Dragstedt in several hundred cases are outstanding. The mortality rate was less than 1 per cent. There was prompt healing of the ulcer, relief of ulcer distress, and negative insulin tolerance tests in 90 per cent, while persisting or recurring ulceration occurred in slightly more than 3 per cent and these occurred in that group which he considered represented incomplete vagotomy because of the positive insulin tolerance test. Undesirable side effects he found to be only transient and easily controlled. Only 3 failures resulted in Bancroft's series of 34 vagotomies, but Gardner and Hart reported symptoms of gastric retention to be of major importance in over one-third of their cases, necessitating a secondary operation in 14.3 per cent of the total. From the Mayo Clinic, Walters reported troublesome motility effects occurring with equal frequency among the positive and negative insulin tolerance groups but, surprisingly enough, nearly all the cases of recurrent ulceration or persistence of ulcer occurred in the group with negative tests. In the Lahey Clinic series there were instances of recurrent ulcers and recurrent hemorrhages. Smithwick recently showed a rather surprising effect of sympathectomy upon gastric acidity in the presence of peptic ulcer. Hypertensive patients with coincidental peptic ulcers who underwent thoracolumbar sympathectomies did not show the anticipated rise in hydrochloric acid levels which were expected to follow from an unopposed vagal effect. On the contrary, they exhibited a diminshed level of hydrochloric

response to test stimuli. Smithwick discovered the records of 3 such sympathectomized patients who subsequently were subjected to emergency gastrectomy for bleeding ulcer when only the gastric antrum was removed. These patients exhibited postoperatively an achlorhydria which Smithwick proposed as the physiological result of the removal of only the gastric antrum and sympathetic nerves with the vagus still intact.

EVALUATION OF VAGOTOMY

Efforts to determine the place of vagotomy have led to a reevaluation of the results obtained by other surgical procedures in the treatment of peptic ulcers. In an analysis of the elective surgical procedures performed for peptic ulcer in our own group, there are 17 subtotal gastric resections and 1 gastroenterostomy, and the latter operation was performed for symptoms of obstruction by a relatively inactive cicatrizing duodenal ulcer. In the gastric resections we had 1 death, a mortality rate of 6 per cent, which was the result of pulmonary embolism. One stomal ulcer occurred which necessitated a second operation. This resulted from an exclusion type of resection. We do not hesitate to exclude the ulcer when it is in such a position as to be unduly hazardous to remove, but, of course, the antral mucosa should be removed when any part of the pyloric margin of the stomach is permitted to remain. Wangensteen has reported a series of nearly 100 such operations without the occurrence of a stomal ulcer. Malfunctioning stomas were troublesome in 2 of our cases and there was a mild dumping syndrome in yet another. In all, the final end results were considered to be completely satisfactory in only 82 per cent of the total. These results are fairly similar to a much larger series reported by Gardner and Hart whose operative mortality was 8.9 per cent but when excluding the emergency operations for hemorrhage, the corrected mortality was 6.6 per cent. Five per cent had recurrent ulceration and 6 per cent had postgastrectomy syndromes consisting of fullness after meals, regurgitation of food, nervousness, weakness, dizziness, and inability to regain strength or weight. Similar analyses were made by F. B. St. John showing 85 per cent satisfactory results.

In New Orleans, the mortality rates for gastric resection for peptic ulcer as furnished by the record librarians in the following institutions are: Ochsner Clinic, 4 per cent; Touro Infirmary, 11 per cent; Charity Hospital, 11 per cent; Baptist Hospital, 7.6 per cent. The lower mortality rate for the nerve resection is very appealing when contrasted with the higher mortality rates for resection even at the expense of motility disturbances in a minority of patients. The incidence of stomal ulcer following gastric resections is reported as 5 per cent from the Lahey Clinic and as the same figure from Duke University Hospital. In both institutions an antecolic, Hoffmeister technic was the procedure usually employed. The Ochsner Clinic has the enviable record of 119 gastric resections with no incidence of marginal ulcer. Ochsner contends that when an adequate subtotal resection is performed and that when a short-loop, posterior gastroenterostomy is employed that the decreased vulnerability of the jejunal mucosa causes minimal trouble with stomal ulcer. Nevertheless, when one compares the average incidence of recurring marginal ulceration following gastric resection with the persisting or recurring ulceration after vagotomy, the results seem fairly close. The distressing symptoms of the postgastrectomy syndrome are balanced against the undesirable side effects which follow the nerve operation which occur in about the same ratio. Thus from the nerve operation, the mortality rate is low, the relief of ulcer pain is dramatic, the incidence of recurrent ulceration is about the same as that following gastrectomy, but a big disadvantage is the high incidence of gastric retention which requires a secondary operation in a fair percentage of cases. Dragstedt contends that the prevention of overdistention of the stomach in the early postoperative period by use of Wangensteen suction reduced this undesirable side effect to a minimum.

Where, then, should vagotomy be placed in the treatment of peptic ulcer? It is rather generally accepted that it is the treatment of choice in marginal ulcer following gastric resection, and concurring in this opinion are Ochsner, Lahey, Walters, Hart, as well as the more ardent supporters of vagotomy. In the treatment of such cases, Priestly reported only 1 poor result in a series of 16 cases of marginal ulcer following resection and 21 cases of gastrojejunal ulcer after gastrojejunostomy. Walters considers that the resection of the stomach and not the nerve is the procedure of choice in all other situations. It is less generally accepted that the nerve operation alone is dangerous in the treatment of gastric ulcer because of the high incidence of malignancy in these ulcers, but Crile favors the nerve operation for those gastric ulcers which are so high in the stomach that their danger of malignancy is less than the added risk of mortality of near total gastric resection which would be required for their removal. Moore considers young and middle aged patients, with chronic ulcers which occur under stress and strain and are only transiently relieved by food and alkali, as the ideal subjects for vagus resection. Hart is of the opinion that there is such a high incidence of gastric retention following vagotomy alone that it should probably never be used without a concomitant gastroenterostomy, whereas Lahey is opposed to gastroenterostomy because of the possibility of gastrojejunal ulceration if the acidity and motility are restored with the passage of time. Dragstedt, on the other hand, states that no other surgical method has obtained such good results in the treatment of gastric and duodenal ulceration, with and without gastroenterostomy, and on his service gastric vagotomy has replaced all other types of surgery in severe and intractable peptic ulcers. In our own cases, we, as well as our patients, have been satisfied, and the vagus resection operation compares favorably with the gastric resection series; however, the number is too small to be statistically of much value. If time proves Moore's observations to be correct in that the stomach becomes autonomous while still being relieved from cephalic influence, and if gastrojejunal ulceration does not take place with this return toward a normal physiology, then the operation should gain wider application. Until then we should not perform vagus resection promiscuously, nor should we consider that it alters the rigid indications for surgical intervention in the treatment of peptic ulcer.

CONCLUSIONS

1. The majority of the leading surgeons in New Orleans consider vagotomy to be a poor operation for peptic ulcer, except in gastrojejunal ulceration after adequate subtotal gastric resection, where vagotomy is rather generally accepted to be the operation of choice.

2. There is no truly ideal operation for peptic ulcer. After vagotomy there are a significant number of distressing hypomotility symptoms, recurrent or persistent ulceration, and diarrhea. After subtotal gastric resection there is a higher mortality rate, and approximately 10 per cent to 20 per cent with symptoms due to postgastrectomy syndrome, "dumping syndrome", inability to gain weight, or marginal ulceration.

3. In the group of vagotomy cases collected from various hospitals in New Orleans, less than 75 per cent were judged as satisfactory results. Our own vagotomy series, however, compared very favorably with the results of our larger series of gastric resections.

REFERENCES

1. Gardner, C. E., Jr., and Hart, Deryl: The surgical treatment of peptic ulcer. Ann. Surg., 127:1056, 1948.

2. Lahey, F. H.: Diagnosis and treatment of peptic ulcer. Postgrad. Med., 3:342, 1948.

3. Allen, A. W., and Welch, C. E.: Subtotal gastrectomy for duodenal ulcer. Ann. Surg., 124:688, 1946.

4. Walters, W., and Neibling, H. A.: Vagus nerve resection for peptic ulcer. Postgrad. Med., 4:6, 1948.

5. Moore, F. D., Chapman, W. P., Schulz, M. D., and Jones, C. M.: Transdiaphragmatic resection of the vagus nerves for peptic ulcer. New England J. Med., 234:241, 1946.

6. Camp, E. H. and Dragstedt, L. R.: Gastric vagotomy in peptic ulcer. Postgrad. Med., 3:8, 1948.

7. St. John, F. B., *et al*: Results following subtotal gastrectomy for duodenal and gastric ulcer. Ann. Surg., 128:3, 1948.

8. Smithwick, R. H.: Surgery of the autonomic nervous system. New England J. Med., 236:662, 1947.

9. Bancroft, F. W.: In discussion of "The surgical

treatment of peptic ulcer". Ann. Surg., 127:1056, 1948.
10. Grimson, K. S., *et al*: The effect of transthoracic vagotomy upon the function of the stomach and upon the early clinical course of patients with peptic ulcer. South. M. J., 39:460, 1946.
11. Dragstedt, L. R.: Some physiological principles in surgery of the stomach. Canad. M. A. J., 56:133, 1947.
12. Stein, I. F., Jr., and Meyer, Karl A.: Studies on vagotomy in the treatment of peptic ulcer. Surg. Gynec. & Obst., 86:473, 1948; Surg. Gynec. & Obst., 87:188, 1948.
13. Ochsner, Alton: Personal Communication.
14. Penick, R. M., Jr.: Personal Communication.
15. Mahorner, H. R.: Personal Communication.
16. O'Neil, L. J.: Personal Communication.
17. Smithwick, R. H.: Peptic ulcer with particular reference to the effects of various surgical procedures upon gastric acidity. Paper read before The New Orleans Graduate Medical Assembly, March 10, 1949.

A GASTROENTEROLOGICAL EVALUATION OF VAGOTOMY IN THE TREATMENT OF ULCER*

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NEW ORLEANS

This review is presented to evaluate a method of surgical treatment for peptic ulcer currently in vogue, which involves resection or extirpation of the vagus nerves by either thoracic or abdominal approach. Although firsthand knowledge of the results of this operation is extremely limited in this medical center, a summation of existing knowledge on vagotomy serves a useful purpose. We are enabled to re-evaluate a live gastroenterological subject—the optimal means of treating ulcer and of appreciating the physiology of the vagus nerve as related to this disease.

ANATOMY

The vagi arise from the medulla and pursue the longest course of any of the cranial nerves down to the esophageal plexus. Below this, the right nerve forms a single cord which courses along the back of the esophagus through the diaphragm to be distributed to the posterior and inferior surfaces of the stomach. The left nerve continues to the anterosuperior surface of the stomach and terminates in the fundus and lesser curvature. These preganglionic, parasympathetic vagus fibers terminate in the plexus of Auerbach situated in the stomach's muscular coat. Interest in surgical division of the vagi has led to further studies of the course of these nerves. Bradley et al dissected 100 esophagi. In 92, one of three situations existed; in 64 two trunks were formed between the hiatus and 6 cm. above the diaphragm as in the classical anatomic description; in 7, there was a large plexus which formed two trunks at the hiatus; and in 21, long trunks existed for a distance of 6 cm. above the diaphragm. In 8 or 10 per cent, there was no constant or uniform course, and multiple branches existed. Chamberlain and Winship dissected 50 esophagi and found in 12 cases, or 24 per cent, that there existed a complex arrangement with two or more nerve trunks representing each vagus nerve. Bradley and co-workers found 8 per cent of cases with many small branches.

EFFECTS OF VAGOTOMY

A fairly good correlation exists between the results of operation in animal and man. McSwiney found that the stomach nerves affect either pure tonus change or affect the rhythmic movements of this organ. The totally denervated stomach may function adequately. The physiologic changes after vagotomy are many. The vagi relax the cardiac sphincter in advance of peristalsis and exert a tonic effect on posture of the stomach. When the nerves are cut, achalasia develops, the stomach loses tone and becomes more readily distended with air. In dogs the tonic type of hunger contraction is lost. The vagus is inhibitory for the pylorus. There is questionable small intestine action and no demonstrable effect on the colon. The secretory effect of vagotomy is to abolish the cephalic or psychic phase of gastric secretion. Moore *et al* find in man a decrease of stomach motility with reduced acid in the resting unstimulated stomach. There is no change in sensation and no change in sensory threshold to balloon distention of the esophagus, stomach, duodenum, jejunum, and ileum. Nausea, vomiting, and epigastric fullness may still be experienced. Motility changes below the pylorus are transient and inconstant. The insulin test is the most reliable stimulus to demonstrate physiologic effects of vagot-

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omy on the stomach. Griswold found that the average night secretion in twelve hours was 1300 cc. before, and 270 cc. after vagotomy. Andrus was able to study a patient with a gastrostomy opening. He found that anger and resentment produced a typical vascular response with marked reddening of the mucosa. After vagotomy painful emotion, strong enough to bring a flush to the face, no longer affected the gastric mucosa. A study of reported cases leads to the conclusion that complete vagotomy promptly relieves ulcer pain. The means by which this takes place is not too clear. This effect is not due to section of the few afferent sensory vagus fibers, since they mediate reflexes and are not able to transmit pain. Relief of ulcer pain is also not directly related to reduction of acidity which usually follows this operation, since in some cases, pain relief occurs without any lowering of acidity. Pain relief may be due to diminished or absent peristaltic activity. Grimson introduced free acid into human stomachs without producing pain.

A consideration and evaluation of the results of vagotomy in man must of necessity be a slow process. Observation over a ten to twenty year period will give an accurate appraisal of the duration of relief and the relapse rate. Results of animal experimentation are important and will tend to forecast the future of vagotomy. Ferguson performed vagotomy on 10 monkeys. Cardiospasm was found in all and gastric hypotonia with delay in the onward passage of solid food. The acid tide was not lowered in 6. Two showed mucosal erosions at postmortem examination. Several workers studied vagotomy in conjunction with the Mann-Williamson operation. Preliminary vagotomy did not protect against these ulcers. After vagotomy, rats on a stock diet died in ten days from blood impacted in the stomach. The animal becomes an invalid and dies unless managed very carefully. Shapiro and Berg did subtotal gastrectomy and vagotomy in dogs with only temporary reduction in gastric acidity. They concluded that the stomach has compensating mechanisms. Beazell and Ivy after vagotomy on rabbits found that on a rough diet 66 per cent develop ulcers. On a smoother diet the incidence was 15 per cent. He believed that these ulcers resulted from mechanical trauma associated with gastric stasis. Alvarez found that half the rabbits after this operation developed diarrhea.

INSULIN TEST

The insulin test has been devised to measure completeness of vagotomy. Hollander performs this test by giving a Sippy diet the day before the test. No food is given after 7 p. m. and no fluid after midnight. A Levin tube is passed and the patient instructed to expectorate all saliva. Fifteen units of regular insulin are given intravenously. Every fifteen minutes thereafter the stomach is completely aspirated and a record kept of the time, volume, gross presence of mucus and bile, and titration of acidity. Every thirty minutes a blood sugar is run. Aspirations are kept up for two hours. At the Mayo Clinic 0.3 unit of regular insulin is given intravenously for each kilo of body weight; a 70 kilo patient would thus be given 21 units. An insulin test is considered negative when, with a blood sugar below 40, and adequate secretory cells there is no increase in free gastric acid. Complete vagal interruption produces a negative test. A positive insulin test occurs when the response to this procedure is an appreciable rise in free acid. The hypoglycemia acts like a central nervous system stimulus, probably acting on medullary centers, causing secretory impulses to be transmitted by way of the vagus nerves to the gastric mucosa. A fall in blood sugar to 50 mg. is usually sufficient to cause a rise in gastric acid. Glucose, intravenously, atropine, or vagotomy counteract this effect. The gastric juice secreted under these circumstances has the characteristics of pure vagal juice, a very high acid titre, stronger pepsin content than after histamine, and a greater amount of visible mucus. All secretory glands of the stomach are activated by the insulin reaction and hypoglycemia.

The test has proved accurate in dogs in which three types of gastric pouches were studied: the Heidenhain with no vagal in-

terruption, Pavlov with a small percentage of vagal fibers intact, and the vagal with all the vagus fibers severed. The response to sham feeding and the insulin test were studied. Hollander concluded that the test is not quantitative and does not indicate the amount of uncut nerve tissue. A negative secretory test may be due to several factors: (1) complete vagal section; (2) inadequate hypoglycemia even in the presence of some intact vagal fibers; (3) temporary achlorhydria even to histamine; (4) intestinal regurgitation. He applied the test to 43 ulcer cases. All but one gave rise to increased acid secretion. Postoperatively the test was applied to 21 patients with 11, or 52 per cent, giving a negative test. Opinion is divided as to the usefulness of the insulin test in man. Will it determine the completeness of vagotomy, the prognosis after operation, and which cases are future candidates for vagotomy? Waltman Walters found that the reduction in gastric acid and motility bore no relationship to the insulin test. Recurrences for the most part occurred in the insulin negative cases. In 10 cases with suspected recurrence after vagotomy, only one had a positive test. Moore *et al* found that complete vagotomy abolished the acid response to insulin induced hypoglycemia in the early postoperative phase. Harkins and Hooker did the test on 17 patients postoperatively; of 3 cases reacting to the test, 2 were reoperated and ulcers were found.

CHOICE OF APPROACH

It has been realized for many years that complete transsection of both nerves was necessary to bring about the effects of this operation. The situation has not changed since 1892 when Krehl stated that transthoracic vagotomy in animals produced so much gastric stasis that dogs could hardly be kept alive while those who cut all fibers below the diaphragm saw no change in their animals. The few fibers which were left must have taken over all the functions of the two big nerves.

Because of anatomic variations complete vagotomy will be difficult or impossible in from 8 to 24 per cent of cases by either approach. Weinstein, Colp, *et al* report a case autopsied after the surgeon believed a complete vagotomy had been performed and found that only 60 to 80 per cent of the fibers had been interrupted.

Transthoracic vagotomy has been entirely abandoned by Dragstedt, the surgeon who first gave renewed impetus to the study of effects of vagotomy. He concluded that chest pain postoperatively is annoying; that the transthoracic route does not permit abdominal exploration to confirm the diagnosis, and does not permit simultaneous abdominal operation. Grimson abandoned this procedure because 10 patients required drainage operations later. Because of anatomic variations Chamberlain and Winship question the practicability of the transabdominal approach. Colp and Klingenstein in the treatment of recurring gastrojejunal ulcers use the transthoracic approach.

The abdominal approach is more widely employed at the present time. It is best for the treatment of duodenal ulcer because other operations may be combined with it during laparatomy. It also becomes necessary when gastric ulcer requires opinion as to possible malignancy of the lesion.

CLINICAL RESULTS

Cliinical results of vagotomy to date vary. Evaluation of vagotomy is difficult because there is no uniform selection of cases, because of the natural relapse rate of ulcer patients, and because of the different technics of vagotomy performed. It is also difficult to evaluate vagotomy because it is so frequently combined with other satisfactory, accepted surgical methods of treating ulcer, such as gastroenterostomy and gastric resection.

Dragstedt reviewed the results of 450 patients operated upon by him. During the period between January 1943 to mid-1945, 80 patients had transthoracic vagotomy. This was abandoned because of residual chest pain and inability to explore and perform simultaneous abdominal operation. From 1945 to mid-1946 vagotomy and gastroenterostomy were performed on all duodenal ulcers. The results were good. Since

1946 he has decided on the basis of pyloric stenosis whether or not anastomosis was necessary. Vagotomy alone was done on 202 patients; 168 had vagotomy plus gastroenterostomy. There were 4 deaths. Three hundred and forty insulin tests showed complete vagus section. The ulcer recurred in 8. He states that he was able to control the side effects of the operation.

Certain unfavorable results are reported. Warren reports a patient who developed pain three weeks after vagotomy and was found to have retained 80 per cent of the barium meal. The stomach was resected and showed two gastric ulcers, one chronic and one acute. Grimson, Baylin, *et al* operated on 57 ulcers which were refractory. Secondary gastrenterostomy was necessary in 5 of the 41 patients originally treated by vagotomy alone. Eleven had persistent fullness after meals, 10 had foul eructations, 5 had lasting colicky pain, 1 had serious diarrhea, and 11 had continuous or periodic looseness of stools. Nine patients of 32 were completely satisfied with the operation. Later, reporting results in 104 patients in a 4 years, Grimson and Baylin concluded that vagotomy alone is not a satisfactory treatment for ulcer. One out of 4 cases required later gastroenterostomy. One out of 10 had ulcer pain, and in 1 out of 25 there was definite persistence or recurrence. Of this group only 23 of 49 patients were satisfied with the end results.

Moore first reported the results of the operation to be good in 87 per cent of his cases and poor in 5 patients, and 5 other patients had severe side effects. Later he stated that 11 per cent of the cases recurred. There were also postoperatively patients with minor symptoms, who showed x-ray evidence of ulcer. He is still following the cases very carefully. Walters in 1948 stated that we must allow five to ten years to elapse before finally judging results of this operation. He finds that with vagotomy plus gastroenterostomy the results are not much better than that of gastroenterostomy alone. In his experience 35 per cent of unsatisfactory results followed vagotomy alone. Aaron reported 2500 cases reviewed recently by the American Gastroenterologic Association with a mortality of 1.7 per cent. With vagotomy alone there was relief of pain in 85 to 90 per cent and recurrence of symptoms in 15 to 20 per cent. With resection plus vagotomy 84 per cent gave satisfactory results.

The question must be answered: How long do these effects persist after vagotomy? Grimson *et al* state that the effect on motility and on acidity persist up to four years. A few patients at two to four years had a negative insulin test change to a positive. Moore *et al*, however, found that one year after operation gastric motility in the resting state studied by kymograph returned to normal levels. Atony as seen by the fluoroscope was gone in three to nine months. Secretory changes, as tested by multiple quantitative overnight determinations of gastric contents, usually returned to normal in six to twelve months. The work of Vanzant in animals has never been disproved. Four of the 8 dogs previously vagotomized by Hartzell were studied two and a half years after operation. There was a return of normal acid values.

MEDICAL TREATMENT OR SURGERY

The great majority of ulcer cases respond to medical therapy. It behooves the gastroenterologist to study the cause of failure of the usual medical regime very carefully. I have treated apparently classical duodenal ulcer patients whose pain continued until food allergy was discovered and a milk free ulcer diet prescribed. Other patients frequently delude their medical advisers and continue smoking surreptitiously while on ulcer treatment. In other cases ulcer symptoms continue until psychogenic and psychosomatic factors are discovered and controlled. Patients must always receive adequate medical treatment before surgery is contemplated. The problem today is the comparatively small, difficult group of cases who seem to have an ulcer diathesis with marked hyperacidity, continuous interdigestive hypersecretion, and obvious autonomic and psychogenic imbalance. They do badly after the usual operations for ulcer. This is the group on which va-

gotomy should be tried. Until adequate time has passed and the results in the 11,000 vagotomies already performed are known, this operation had best be reserved for the treatment of the following special ulcer problems:

1. Gastric ulcer high up near the esophagus, unless malignant, because of the high mortality after resection.
2. Duodenal ulcer patients who need surgery because of pyloric obstruction in which vagotomy may be performed simultaneously.
3. In recurrent gastrojejunal ulcer in which vagotomy may prove to be the operation of choice.
4. In so-called ulcer diathesis when conventional methods of treatment have failed.

SUMMARY AND CONCLUSIONS

1. The duration of the beneficial physiological effects following vagotomy is not known at this time.
2. A final evaluation of vagotomy in the treatment of ulcer can be made only if this operation is performed alone.
3. Approximitely 10 per cent of patients will have multiple divisions of the vagi above the diaphragm which make the operation of complete section difficult or impossible.
4. The operation has lowered night secretion, lowered gastric acidity, and relieved ulcer pain.
5. Stimulation of gastric acidity by insulin induced hypoglycemia offers a promising physiological tool.
6. The use of vagotomy should be limited at this time to selected cases.
7. The fate of the 11,000 patients already vagotomized should be carefully followed for ten to twenty years, to determine the final results of the operation.

REFERENCES

1. Bradley, W. F., Small, J. T., Wilson, J. W., and Walters, W.: Anatomic considerations of gastric neurectomy, J. A. M. A., 133:459, 1947.
2. Chamberlain, J. A., Winship, T.: Anatomic variations of the vagus nerves—Their significance in vagus neurectomy, Surgery, 22:1, 1947.
3. Walters, Waltman, Neibling, H. A., Brownson, B. C., and Phillips, S. K.: Scientific Exhibit, Section on Surgery, American Medical Assn., June 1948.
4. McSwiney, B. A.: Innervation of the stomach, Physiol. Rev., 11:478, 1931.
5. Ivy, A. C.: Discussion, J. A. M. A. in press.
6. Moore, F. D., Chapman, W. P., Schulz, M. D., and Jones, C. M.: Resection of the vagus nerves in peptic ulcer: Physiologic effects and clinical results, with a report of two years' experience, J. A. M. A., 133:741, 1947.
7. Griswold, R. A.: Discussion, Ann. Surg., 126:15, 1947.
8. Andrus, W. D.: Discussion, J. A. M. A., 133:741, 1947.
9. Alvarez, W. C.: Sixty years of vagotomy, Gastroenterology, 10:413, 1948.
10. Grimson, K. S., Baylin, G. J., Taylor, H. M., Hesser, F. H., and Rundles, R. W.: Transthoracic vagotomy, the effect in 57 patients with peptic ulcer and the clinical limitations, J. A. M. A., 134:925, 1947.
11. Ruffin, J. M., Grimson, K. S., and Smith, R. C.: The effect of transthoracic vagotomy upon the clinical course of patients with peptic ulcer, Gastroenterology, 7:599, 1946.
12. Ferguson, J. H.: Effects of vagotomy on the gastric functions of monkeys, Surg. Gynec. & Obst., 62:689, 1936.
13. Oliver, James V.: Effect of vagotomy on the development of the Mann-Williamson ulcer in the dog, Arch. Surg., 55:180, 1947.
14. Saltzstein, H. C., Sandweiss, D. J., Hammer, J. M., Hill, E. J., and Vandenberg, H. J., Jr.: Effect of vagotomy on Mann-Williamson ulcers in dogs, Arch. Surg., 55:130, 1947.
15. Komarov, S. A.: Discussion, Gastroenterology, 7:615, 1946.
16. Shapiro, P. F., and Berg, C. N.: Return of gastric acidity after subtotal gastrectomy and double vagotomy, Arch. Surg., 28:160, 1934.
17. Beazell, J. M., and Ivy, A. C.: Chronic gastric ulcer following bilateral vagotomy in the rabbit and the dog, Arch. Path., 22:213, 1936.
18. Alvarez, W. C., Hosoi, K., Overgard, A., and Ascanio, H.: The effects of degenerative section of the vagi and the splanchnics on the digestive tract, Am. J. Physiol., 90:631, 1929.
19. Hollander, Franklin: The insulin test for the presence of intact nerve fibers after vagal operations for peptic ulcer, Gastroenterology, 7:607, 1946.
20. Jemerin, E. E., Hollander, F., and Weinstein, V. A.: A comparison of insulin and food as stimuli for the differentiation of vagal and non-vagal gastric pouches, Gastroenterology, 1:500, 1943.
21. Moore, F. D., Chapman, W. P., Schultz, M. D., and Jones, C. M.: Resection of vague nerves in peptic ulcer, J. A. M. A., 133:741, 1947.
22. Harkins, H. H., and Hooker, D. H.: Vagotomy for peptic ulcer, Surgery, 22:239, 1947.
23. Dragstedt, Lester R.: Discussion, J. A. M. A.: in press.
24. Weinstein, V. A., Colp, R., Hollander, F., Jemerin, E. E.: Vagotomy in therapy of peptic ulcer. Surg., Gynec., & Obst., 99:297, 1944.
25. Winklestein, A.: Discussion, Gastroenterology, 7:615, 1946.
26. Warren, R.: Experiences with vagotomy for peptic ulcer with report of an unsuccessful case, Surgery, 22:246, 1947.
27. Grimson, K. S., Baylin, G. J., Taylor, H. M., Hesser, F. H., and Rundles, R. W.: Clinical evaluation of complications observed after transthoracic vagotomy. Arch Surg., 55:175, 1947.
28. Moore, F. D.: Resection of the vagus nerves for ulcer: An interim evaluation. Arch. Surg., 55:164, 1947.
29. Moore, F. D.: Discussion, J. A. M. A.: In press.
30. Aaron, A. H.: Newer aspects of the treatment of ulcer, J. A. M. A.: In press.
31. Moore, F. D., Chapman, W. P., Schulz, M. D., and Jones, C. M.: Resection of vagus nerves in peptic ulcer: Physiologic effects and clinical results, with a report of two years' experience, J. A. M. A., 133:741, 1947.
32. Vanzant, F. R.: Late effects of section of vagus nerves on gastric acidity, Am. J. Physiol., 99:375, 1932.

THE PRESENT STATUS OF ANALGESIA AND ANESTHESIA IN OBSTETRICS*

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NEW ORLEANS

The ideal agent to employ routinely in order to obtain analgesia and/or anesthesia in obstetrics has yet to be found. Principles suggested by Fluhman[1] in 1940 hold true today:

1. The ideal agent must alleviate suffering, not interfere with the progress of labor and be safe for the baby.

2. One must be familiar with the agent and know its contraindications.

3. One must know the dosage and optimum time for administration.

These principles may be applied to and utilized in the administration, as well as the critique, of old and new methods. Nonadherence accounts for the failures and pitfalls in the hands of the profession at large. Where one drug or combination of drugs is successful in one section of the country, the same drug may be discarded or rarely used in another section.

HISTORICAL NOTES

In commemoration of the "Introduction of Anesthesia", Watson[2] relates how Sir Alexander Simpson, a British physician, used ether, for the first time in obstetrics, on January 19, 1847. The anesthetic was given to a patient whose labor was terminated by version extraction. On November 4, 1847 chloroform was first administered. Almost ommediately, controversy was stirred up in this country. The arguments were based on "alleged indecencies committed by patients", plus the issue of whether or not it was right and proper to relieve a woman of the pains of labor by general anesthesia, "against nature".

The general use of anesthesia in obstetrics which soon spread through Scotland, France, England, Germany, and in a few months to America, was soon followed by an increase in maternal and infant mortality. In 1900 Murray of Edinburgh[3] found in his studies, that this increase in mortality was due chiefly to the *misuse* of anesthesia. Interference was limited prior to 1847 because it increased the "conscious suffering of the patient".

ANALGESIC AND ANESTHETIC AGENTS

A brief review of some of the more prominent drugs employed during the past ten years will be discussed. In this manner we can evaluate from large patient series what the results depict.

I. *Morphine Sulfate*: Probably used more than any other drug. It is fast acting, produces pain relief and unless given in very large doses, does not interfere with labor. Narcosis will be present to some degree in the infant if it is administered within three to four hours of delivery. Doses range from 1/6 to 1/4 grain.

II. *Morphine-Scopolamine*: In 1902, Von Steinbuckel[3] used this combination to popularize what was to be known later as "Twilight Sleep". This form of analgesia enjoyed a long and violent popularity. Many babies were lost in deep narcosis. However, the principle of the combination is still used in some forms today, and is of great value if if the dosage is intelligently and carefully administered. Morphine and scopolamine often relax a patient in long, tedious, exhaustive labors, only to have her awaken refreshed and capable of a happy termination. Narcosis and asphyxia may be more profound in the infant than when morphine is used alone. The time element must be closely guarded. Adair and Pearl[4] found by placing a rubber bag in uteri of multipara, five to seven days postpartum, and activating uterine contractions with ergonovine (.2 to .4 milligram doses) that morphine, gr. 1/4, or morphine, gr. 1/4, combined with atropine 1/150, produced no change in uterine tone. Morphine, gr. 1/4, combined with scopolamine, gr. 1/200,

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1/100 or 1/75, produced more relaxation between contractions.

III. *Heroin*: Lund and Harris[5] employed heroin in 454 patients and concluded that it was good analgesic drug. Heroin produces, in addition to relief from pain, euphoria and lack of depression. There was a 3 to 5 per cent increase in asphyxia neonatorum but less so than with morphine. Your essayist had an opportunity to use heroin in 1934 and is in complete agreement. It is regretable that this opiate is unobtainable.

IV. *Demerol. Demerol - Scopolamine*: Alone or in combination, demoral has proved to be a good analgesic drug. Its combination with scopolamine produces deeper analgesia with amnesia.

Although it would appear that demerol is less toxic to the infant than morphine, one must not be lulled into a false sense of security. It is still an opiate in principle and capable of producing severe reaction in the baby. Irving[6] observed only two complications in 2446 patients. The popular administration begins with a dose of demerol, 100 milligrams, with scopolamine, gr. 1/150, when labor is well established, and cervical dilatation has begun. This may be given intravenously although its effect is very satisfactory intramuscularly. Schade[7] uses this method with terminal pudenal block. His results are excellent. Should additional analgesia be desired repeated doses of 100 milligrams or less may be administered, with or without scopolamine. If amnesia alone is desired, scopolamine alone may be repeated in 1/200 grain doses. The effect of demerol on the uterus is similar to that of morphine, allowing a patient to relax, and labor to progress to a normal termination. If terminal inhalation anesthesia is used, this combination is a desirable preanesthetic medication, requiring less gas, with easier induction and a dry bronchial tree.

V. *Dolophine*: A new drug proved disappointing in Lund's[15] study. Compound No. 10720 gave fair to good results in two-thirds of his series. No toxic reactions were observed.

VI. *Barbiturates*: Everyone is familiar with the barbiturates which are administered orally. Each of many varieties has enjoyed popularity. Although the absorption rate, grain dosage, and recovery rates vary somewhat, in reactions of the mother and baby, barbiturates have much in common. In small dosage mild hypnosis is achieved. To obtain amnesia, large doses are necessary and to obtain analgesia or anesthesia via the oral route, near lethal dosage must be employed. The critical dose as far as the baby is concerned is far less. Patient excitement may be troublesome. If given near the delivery hour hypnosis and asphyxia in the infant is usually assured. In combination with morphine or demerol-scopolamine analgesia very small doses of oral barbiturates are helpful in relaxation and alleviation of apprehension.

Irving[6] reported 14,676 patients in whom pentobarbital was administered. Respiratory accidents occurred 1.75 times more than when seconal or amytal was the agent. Pentobarbital in oral doses of 3 grains produces no change in uterine activity except to lengthen the interval of contractions. Barbiturates have some value if rest is desired in lengthy and slow-progressing labors.

VII. *Intravenous Analgesia*: *A. Sodium Pentothal.* Mazzola[8] (one of many investigators) utilized this agent in 300 deliveries. The dose averaged between 0.89 and 1.40 grams, intravenously. Delivery occurred in thirty-eight to seventy-one minutes. In cesarean section he warns that the baby should be delivered in five minutes after the injection. The safety factor is questioned.

This is a rapid acting, comparatively quickly detoxified drug. It bears all of the general objections to barbiturates. In cesarean section under local or spinal, this agent is ideal if used to relax the patient *after* the baby has been delivered.

B. Evipal—Similar in use to pentothal sodium.

C. Vinbarbital Sodium (*Delvinal*). Kohl[9], Lewis and Boddie[10] and Lewis[11] have used this type of analgesia in 3150 patients.

The average dose consisted of 9 grains orally, with scopolamine gr. 1/150 during labor. Scopolamine was repeated when necessary. For delivery, 8-20 grains were administered intravenously. Their operative rate was 53 to 57.5 per cent. It is interesting to note that 11.6 per cent to 18.9 per cent of the infants were asphyxiated to some degree. Postpartum, the mothers slept from one to eighteen hours, often alarming their families.

D. Dial Urethane. A dose of 4 cc. intravenously at 2 cms. dilatation was used by Van Del[12] in 2,000 deliveries. Morphine, grs. 1/8, was given one half hour later. The operative delivery rate was 25.4 per cent. There was no narcosis noted in the infant. This was probably due to the early administration of the drug.

E. Paraldehyde—This agent was given in travenously in 100 patients by Gardner and Sage[14], in doses of 0.7 to 1 cc. of a 10 per cent solution per kilogram body weight. Doses averaged between 36 to 150 cc. Analgesia was of brief duration, arm pain, resulted in 44 per cent, cough in 2 per cent and hiccough in 6 per cent of the patients.

VIII. Rectal Analgesia. A. Gwathmey—This type of rectal analgesia enjoyed great popularity in some centers and is still used by a few. The original formula consisted of:

Quinine alkaloid	20 grains
Alcohol	45 mms.
Ether	2½ oz.
Olive Oil qs. ad.	4 oz.

A specialized apparatus is available for introduction of the drug to simplify administration. Variations of the original formula consist of deleting the quinine and substituting mineral oil for olive oil. Many object to the unwieldly procedure. Ether can be detected on the mother's breath soon after the drug is given and almost universally on the baby's breath at delivery. In some mothers it has the undesirable effect of acting as an enema.

B. Evipal has been used rectally in 705 deliveries. (Anderson[16]). There was an operative incidence of 50 per cent. Five per cent of patients were difficult to manage. The dose used was 1½ grams combined with scopolamine, grs. 1/150.

C. Sigmodol—A barbiturate designed for rectal instillation gained popularity in 1939. Schmidt[17] reported 500 cases of which 40 babies delayed crying and 15 were resuscitated. My associates used this type of anesthesia and analgesia for a few years and abandoned it entirely. The chief objections were unpredictable patient behavior and drowsy babies at birth.

D. Paraldehyde—In the hands of Colvin and Bartholomew[18] this type of agent has stood the test for a number of years. Skilled in its use, it meets their requirements. Six to 8 drams in 1.5 cc. benzyl alcohol are given rectally. In 500 deliveries there were 12 per cent sluggish babies.

IX. Analgesic Drugs Combined with Stimulants: A. Caffeine with Pentobarbodital was given by Daro and Stern to 116 patients.[19] Pentobarbital, 7½ to 10 grains, was combined with caffeine in 5 grain doses up to an average of 12 grains. Nacrosis in the infant was evident in 15 per cent of the infants, whereas 23 per cent were affected when caffeine was not employed.

B. Nicotinamide was used in 159 consecutive labors under barbiturate hyocine anesthesia and considerably fewer babies required resuscitation. (Perdue[20])

C. Scopolamine-Apomorphine was given to 500 patients by Hershenson and Brubaker[21] following the initial dose of 3 grains of seconal. Scopolamine, gr. 1/100, apomorphine, gr. 1/100, was given initially, then 1/150 scopolamine and 1/50 of apomorphine was repeated at two hour intervals. Vomiting occurred in 24.5 per cent, excitement in 21 per cent and there was no appreciable increase in blood loss, and no demonstrable depression in either full term or premature babies even when given shortly before delivery.

Further investigation of the use of stimulant drugs may prove of great value if given to counteract an undesired effect of analgesia or an ill-timed dose.

X. Inhalation Anesthesia: A. Ether—

Low in cost, low in mortality, and its ease of administration, has won the general popularity for ether. Deep relaxation for certain operative procedures can be obtained with ether alone or in combination with the popular gases. Used alone the induction may be lengthy, and it is an upper respiratory irritant.

B. Vinethene may be used for procedures not to last over thirty minutes. (Hartman[22]). It has a very short induction stage. Large amounts of oxygen can be given in conjunction with it.

C. Nitrous Oxide popular for early analgesia is undesirable for long or deep anesthesia due to the low oxygen content for an effective mixture.

D. Chloroform—Portable and inexpenpensive, chloroform is used frequently in the home for terminal analgesia. Deep anesthesia is difficult to administer without danger and it is toxic to both liver and heart. Should not be used in toxemias.

E. Ethylene—More relaxation can be obtained than with nitrous oxide. The oxygen content is higher in anesthetic mixture, recovery is rapid, and if premedication has been given, ethylene is a satisfactory terminal anesthetic. For deep relaxation ether usually must be added.

F. Cyclopropane affords adequate anesthesia for all obstetrical procedures.[23] In 1392 patients, Karp and Richardson,[24] noted the following: The patient receives sufficient oxygen; there were no toxic effects on mother or infant. When atropine was given, laryngospasm rarely occurred and good color, free from anoxia, could be maintained. Irritation to the respiratory tract was minimal. This anesthetic, administered by one well trained, is of distinct value for deep and rapid relaxation.

XI. *Spinal Anesthesia* (Saddle Block) This anesthetic, given in a hypertonic solution of glucose, continues to gain popularity, and is one of the newest. Many thousand instances of its use have now been reported with more or less uniform results. Dieckman *et al*[25], in a very carefully controlled series of 719 deliveries observed that "saddle block was a safe procedure in trained hands and one of great value to parturient and fetus". Beck and Ball[26] here in New Orleans, analyzed 966 private cases and arrived at the same conclusion. In our series[27] and in subsequent experience this form of anesthetic has proved applicable in private practice to about 50 per cent of patients. Other drugs are given for early analgesia. Saddle block is used only for terminal analgesia and delivery (last one to three hours of labor). Operative termination is the rule and midforceps rotations are slightly more frequent. We have had no difficulty with the last and there has been no increase in fetal morbidity or mortality from these procedures. Postpartum bladder catheterizations, once, eight hours after delivery, is the rule, but no infections are noted. The most satisfying feature of this anesthetic is to have the baby cry immediately at birth, free from asphyxia.

This anesthetic should not be used alone in deliveries wherein version extraction or breech extraction is the method of termination, or wherein uterine relaxation is of great importance.

Anyone using spinal anesthesia for delivery must be certain of the administrator and the careful observation of his patient. He further must be adept in the use of forceps at the midplane.

XII. *Spinal Anesthesia in Cesarean Section*: Spinal anesthesia in cesarean section may be extremely dangerous if not thoroughly understood and administered by highly trained anesthetists. Since a higher level must be reached, the patient does not sit up as in saddle block, and there is an almost universal drop in blood pressure, early in the procedure. Intravenous fluids should begin as soon as the patient is in position for surgery. Blood pressure determinations should be taken constantly for the first ten minutes. Occasionally vasopressor drugs are necessary, but more commonly the hypotension disappears and the blood pressure level assumes its former peak. Our anesthetists who understand this mechanism have no hesitancy in employing spinal anesthesia. This form of anesthesia has no place in the small institution unless it is so

manned. Certainly the operator should never give his own spinal anesthetics for cesarean sections.

It remains an ideal anesthetic if well managed. From the patient's standpoint, there is less nausea and ileus. Recovery is rapid, blood loss measurably less and the operative field is quiet. In the interest of the fetus, the baby cries often as soon as its head is delivered from the abdominal incision. In private practice and on the Tulane Service at Charity Hospital this anesthesia has been the one of choice for the past three years. There have been no maternal or fetal deaths or near deaths attributable to the procedure. With proper precaution and administration, it is equal to, and combines all the beneficial effects of local infiltration which we have appreciated for years.

XIII. Local Infiltration: A. *In vaginal deliveries* local infiltration can be given either as a pudendal block or as a parasacral block. This form of anesthetic occasionally becomes useful when delivering a patient who presents contraindications for inhalation and spinal anesthesia. The technic is described in any standard text book. Some deaths have been reported from infection and ischiorectal abscesses. These were no doubt due to faulty technic.

B. *In cesarean section*, local infiltration is possible. It is tedious, time consuming and equal in effect to spinal anesthesia. Occasionally it is a necessary procedure where again the patient is no candidate for spinal or inhalation anesthesia. After the baby is born, either morphine and scopolamine may be used for sedation, or some form of intravenous anesthesia of short duration.

CONTRAINDICATIONS

Most of the lack of success as well as the serious complications resulting from the use of obstetrical analgesia and anesthesia is derived from injudicious choice or agent, or its abuse.

Greenhill[23] points out in his review: In 44,894 anesthetics given at Wisconsin General, by well trained anesthetists there were 47 deaths or 1 per 1000. In a series of 988,588 anesthetics using ethylene, nitrous oxide, local cyclopropane, ether, chloroform and spinal, the death rate varied from 0 per 100,000 (31,426, ethylene) to 42 per 100,000 (91,263, spinal). There were 11 per 100,000 (146,970, local). The average was 17 deaths per 100,000. In this he condemns spinal[23]. Certainly in as large a series as this, all types of surgery, on all types of risks were included. One can only criticize the choice of agent, if at all.

Generally speaking, in obstetrics at least, general or spinal anesthesia should be omitted in patients with coronary occlusion, angina pectoris, congestive heart failure, or syphilitic aortitis, for they carry a high mortality. Valvular heart disease, uncomplicated by myocardial involvement, offers little difficulty to any form of anesthetic[3]. In thyrotoxicosis an abundance of oxygen is desirable. In upper respiratory complications at delivery, spinal or local will give the best results. Severe pre-eclamptics or toxemias are usually best delivered over pudendal block. This is also true for patients delivered with acute poliomyelitis and active tuberculosis.

Where cesarean section is necessary and general and spinal anesthesia is contraindicated, local infiltration will suffice.

Short acting intravenous anesthesia agents may be of value in many of the mentioned conditions either alone or in conjunction with local blocks.

COMPLICATIONS IN THE MOTHER

Analgesic drugs will produce marked excitement in many patients if used in large doses. This is particularly true of scopolamine-barbiturate mixtures. The patient's response is unpredictable beforehand and she soon becomes a menace to herself as well as to the obstetrical ward. Constant nurse attendance is mandatory and often unavailable. Extreme excitement can often be relieved only by additional drugs which immediately increase the threat of narcosis in the baby a hundredfold.

Barbiturates lower the prothrombin level in the blood of both mother and baby[28], even in small doses. This is an additional indication for the routine giving of vitamin K to newborns at delivery.

Edema of the uvula and glottis has been reported on 8 occasions since 1945, due to scopolamine[29-30]. Scarification relieved 6, and in 2 tracheotomy was performed to save the lives of those women. Positive skin tests were noted. This is a serious complication which should be remembered, recognized and treated promptly.

Aspiration of stomach contents occurs when inhalation anesthesia is given.[31-32] If solid food is aspirated, massive collapse or a lung abscess may develop. If liquid is aspirated, a pneumonitis will develop. Prior to aspiration, should deep analgesia have been employed, particularly barbiturates, the cough reflex is abolished further complicating the picture. Mendelson[31] reported 2 deaths on the delivery table from aspiration. I know of 1 death at Charity Hospital during the past year. This patient aspirated stomach contents and died before any corrective measures could be instituted.

The underlying cause is delayed gastric activity with prolonged gastric retention during labor. Patients in labor should be restricted to surgical liquids. They should be told beforehand not to eat solid food if labor is suspected. Should emergency surgery become necessary shortly after a full meal has been consumed, gastric lavage should be part of the preoperative preparation. Spinal or local anesthesia lessens the risk. At any cost, every delivery room should be equipped with adequate suction facilities, airways, laryngoscope, and emergency tracheotomy set. When these tragic emergencies arise, time may be the deciding factor between life and death.

COMPLICATIONS IN THE BABY

Every day more attention is drawn to the importance of preventing asphyxia neonatorum. A deeply narcotized baby is a poor exchange for relief of consciousness in its mother. Although there is no measured relationship between asphyxia neonatorum and the type of anesthesia used, prematurity, complications of pregnancy and labor, method of delivery and misuse of analgesic agents all play some part[33]. The longer the anesthetic, the more the baby will absorb, Deftness, dexterity, and knowledge of an obstetric procedure play important roles.

Morphine depresses respiration in the baby and decreases the volume of breathing by lowering the responsiveness of the respiratory center to carbon dioxide produced in the tissue of the body.[34] But even so, carbon dioxide will stimulate the respiratory center.

Barbiturates on the other hand, produce deep narcosis and the governing of respiration by carbon dioxide is abolished. Air is inadequate to support life and excessive increase of carbon dioxide in the blood and lungs excludes oxygen. These babies must be saved only by heroic efforts using oxygen alone. If analgesia is used, the obstetrician must be versed in the use of, and have available, a tracheal catheter and oxygen.

One of the most vicious cycles in obstetrics is that situation wherein a patient in long tedious labor demands maximum sedation. Due to the length of labor she receives more than her share. The complicated labor is finally terminated by operative means and the infant receives both trauma from the sedation and anesthetic, as well as from the operative procedure. Many of these deeply asphyxiated, unfortunate children live subnormal lives.

Although no accurate gauge is available as to how much anoxia a given infant can tolerate, attention is directed repeatedly to the behavior pattern in later life of children so affected. Rosenfield and Bradley[35] studied 673 children between the ages of three and thirteen. (Only children of superior intelligence). A marked increase in abnormal behavior pattern was noted in children who had had difficult resuscitation in infancy or asphyxia illness due to pertussis. The following is impressive:

Behavior	Occurrence in Babies Asphyxiated at Birth	Occurrence in Control
a. Variability in mood	64.3%	40%
b. Hypermotility	85.7%	57%
c. Impulsiveness	75.0%	44%
d. Short Attention Span	89.3%	42%
e. Fluctuant Recall	78.6%	35%
f. Difficulty in Arithmetic	53.6%	22%

Any mother or her physician will find difficulty in rationalizing the justification for heavy sedation if the ultimate result will be a problem child.

A SIMPLE PLAN

A. A patient's behavior in labor begins with preparation during the prenatal period. Few prospective mothers will demand complete oblivion in labor if they know what to expect, if they know the dangers to the baby. They must be taught that the most important item in a pregnancy is a live, uninjured baby. Then they must be assured and reassured that you will present that baby with as little discomfort as is necessary. Assurance, particularly to the primapara is paramount.

B. When labor is established, the obstetrician, (not the nurse or interne), should be present to evaluate the picture and reassure the patient as to her progress. Most patients will complain little if they know what is going on and what to expect in the immediate future.

C. As soon as labor progresses and the tempo of contractions with pain is intensified (with knowledge of the fetal status), demerol, 100 milligrams, combined with scopolamine, grain 1/150, intramuscularly, will suffice to take the "sting" out of the contractions and produce mild amnesia. The patient should be told what to expect from the analgesia. Rarely is more than one repetition necessary. The majority of patients will need only the one dose. If repeated, demerol, 100 milligrams, is given and scopolamine added if amnesia is desired (1/200 gr.). Once in a while for early apprehensiveness, seconal, grains 1½ to 3, may be given. Sedation is withheld or at least limited, if delivery is anticipated within three to four hours.

D. When the cervix becomes dilated to 8 cms. in a primipara or 5 to 6 cms. in a multipara, providing that the presenting part is at or below the level of the ischial spines, saddle block terminal anesthesia is employed. Pontocaine, 10 mg., is given if the duration of one to two hours is desired, and novocaine, 5 mg., if longer analgesia is desired. The average patient coasts in comfort to the stage for delivery, and delivery without further anesthesia is the rule.

E. If, often in multipara, delivery is anticipated in the near future, extremely light ethylene-oxygen anesthesia is administered with contractions and deep induction is administered for delivery. Universally the obstetrician is rewarded with a vigorous, healthy, unharmed baby, who will cry spontaneously.

If complicated operative deliveries, breech extractions and the very occasional version extraction, no anesthesia is begun until the stage is set for delivery. Deep cyclopropane anesthesia will render the task less difficult.

Although one cannot fully subscribe to the psychological approach of Grantley Read[36], there must exist a mode between this and the common practice of forced coma in labor. If one errs, a better result will be the rule if that error is on the conservative side.

To quote Watson[2] once more:

"No honest obstetrician can ever promise to any woman an absolutely painless labor. He can promise to make her labor as painless as possible consistent with safety to herself and to her child. He cannot beforehand accede to a request for some particular form of pain relief. He must reserve to himself the choice of which method he will use and what stage in labor he will use it, according to circumstances."

REFERENCES

1. Fluhman, C. F.: Analgesia and anesthesia in obstetrics, West. J. Surg., 48:361, 1940.

2. Watson, B. P.: Commemorative of introduction of anesthesia, Am. J. Obst. & Gynec., 56:205, 1948.

3. Adair, F. L.: Obstetrics and Gynecology, Lea and Febiger, New York, 2:777, 1940.

4. Adair, F. L., and Pearl, S. H.: Effect of certain sedatives and analgesics on uterine contractions, Am. J. Obst. & Gynec., 35:632, 1938.

5. Lund, C. J., and Harris, J. W.: Use of heroin in labor, Pennsylvania M. J., 46:700, 1943.

6. Irving, F. C.: Advantages and disadvantages of barbiturates in obstetrics, Rhode Island M. J., 28:493, 1945.

7. Schadel, L. M.: A method of obstetrical analgesia and anesthesia, Am. J. Obst. & Gynec., 55:1016, 1948.

8. Mazzola, V. P.: Sodium pentothal anesthesia in major obstetrics and gynecology, Am. J. Obst. & Gynec., 53:207, 1947.

9. Kohl, M. F. F.: Intravenous vinbarbital sodium for obstetrical analgesia, Am. J. Obst. & Gynec., 56:811, 1948.

10. Lewis, M. S., and Boddie, J. B., Jr.: Vinbarbital sodium for obstetrical amnesia, analgesia and anesthesia, South. M. J., 41:820, 1948.

11. Lewis, M. S.: Vinbarbital sodium for obstetrical

amnesia analgesia and anesthesia, Am. J. Obst. & Gynec., 51:395, 1946.

12. VanDel, D. T.: Intravenous dial-urethane in obstetrics, J. Missouri M. A., 35:79, 1938.

13. VanDel, D. T.: Intravenous dial-urethane in obstetrics, J. Missouri M. A., 39:100, 1942.

14. Gardner, H. L., and Sage, E. C.: Intravenous alministration of paraldehyde during labor, Am. J. Obst. & Gynec., 42:467, 1941.

15. Lund, C. J.: Preliminary evaluation of compounds 10720 and 10820 (dolophine), Am. J. Obst. & Gynec., 55:1007, 1948.

16. Anderson, H. E.: Combined evipal and scopolamine analgesia and cyclopropane in obstetrics Am. J. Obst & Gynec., 53:758, 1947.

17. Emmert, F. V., Schmidt, S. G.: Obstetrical analgesia with sigmodol, Journal Lancet, 61:213, 1941.

18. Colvin, E. D., Bartholomew, R. A.: Improvements in paraldehyde method of relief of pain in labor, Am. J. Obst. & Gynec., 35:589, 1938.

19. Daro, A. F., Stern, P. J.: Caffeine as an aid to sodium pentobarbital analgesia in labor, Am. J. Obst. & Gynec., 43:94, 1942.

20. Perdue, J. R.: Use of nicotinic acid as an adjunct in obstetric analgesia, South. M. J., 36:198, 1943.

21. Hershenson, B. B., Brubaker, E. R.: Scopolamine and apomorphine in labor, Am. J. Obst. & Gynec., 53:980, 1947.

22. Hartman, W. F.: Experience with vinethene as an obstetrical anesthetic, Pennsylvania M. Jour., 45:236, 1941.

23. Greenhill, J. P.: Anesthesia in obstetrics, Am. J. Obst. & Gynec., 54:74, 1947.

24. Karp, M., Richardson, G. C.: Technique for administration of cyclopropane in obstetrics, S. Clin. North America, 23:59, 1943.

25. Dieckman, W. J., *et. al.*: Spinal, (saddle block) anesthesia in obstetrics, Am. J. Obst. & Gynec., 55:806, 1948.

26. Beck, M. C., Ball, R. C.: Spinal anesthesia in obstetrics, South. M. J., 41:467, 1948.

27. King, E. L., Dyer, I.: Saddle block in obstetrics, New Orleans M. & S. J., 100:70, 1947.

28. Fitzgerald, J. E., Webster, A.: Obstetrical significance of barbituates and vitamin K, J. A. M. A., 119:1082, 1942.

29. Steinberg, M.: Edema of the glottis and uvula: A reaction to demeral-scopolamine analgesia, Am. J. Obst. & Gynec., 50:542, 1945.

30. Jack, W. W., Taylor, E. S.: Edema of the uvula and glottis associated with demerol-scopolamine analgesia, Am. J. Obst. & Gynec., 56:587, 1948.

31. Mendelson, C. L.: Aspiration of stomach contents into lungs during obstetric anesthesia, Am. J. Obst. & Gynec., 52:191, 1946.

32. Hall, C. C.: Aspiration pneumonitis: An obstetrical hazard, J. A. M. A., 114:728, 1940.

33. Lund, C. J.: Relationship of inhalation anesthesia to asphyxia neonatorium, Am. J. Obst. & Gynec., 43:365, 1942.

34. Henderson, Y.: Use and abuse of barbituates and other narcotics, J. A. M. A., 114:430, 1940.

35. Rosenfield, G. B., Bradley, C.: Childhood behavior sequelae of asphyxia in infancy, Pediatrics, 2:74, 1948.

36. Read, G. B.: Childbirth Without Fear, Harper Bros., N. Y., 1944.

DISCUSSION

W. D. Beacham, M. D. (New Orleans): In the erudite manner which characterizes him, Dr. Dyer has presented an excellent evaluation of the present status of obstetric analgesia and anesthesia. At the outset he very properly recited the fundamentals by which all of the methods must be judged. His review of some of the more prominent drugs employed during the past decade is an example of fairness. Many of us here this afternoon are convinced that demerol is much less hazardous to the infant than is morphine. Some of us do not hesitate to give 50 or even 100 mgm. of demerol to a patient, depending upon her weight, thirty to sixty minutes prior to section under spinal analgesia, although we certainly would not give a similar patient a comparable dose of morphine. His word of caution is well taken, one must always consider the possibility of drug sensitivity and be prepared to counteract it.

Babies in utero, like grown persons, show marked variations in what they can tolerate. Some of them are easily narcotized and traumatized while others are able to withstand large doses of drugs and high degrees of trauma. Excepting those cases with known fetal or maternal complications, there is at present no reliable way of telling in advance just which babies in utero will show a satisfactory drug tolerance. That means that we must be prepared to come to the rescue of any baby that fails to breathe satisfactorily immediately after birth regardless of how little or how much sedation has been employed. All of us well know that it is very important to aspirate the nares, mouth, and oropharynx during the actual delivery. Furthermore, we must have a suitable tracheal catheter so as to aspirate mucus, amniotic fluid, etc., from the pharynx, and if indicated, the trachea, so that the baby will have an unobstructed tract for the passage of oxygen. A first class resuscitation machine is so constructed that it protects the baby from dangerous degrees of positive or negative pressure and is indeed often life saving.

In our practice, we clear the air passages and administer oxygen by the resuscitator or inhalator part of the machine depending upon the respiratory activity of the individual baby. We caution against the loss of valuable time before getting oxygen into the baby's lungs. Furthermore, we again repeat the fact that many babies have a low trauma threshold; consequently, too vigorous attempts at resuscitation by uncontrolled pressures may defeat the purpose for which they are employed.

Routinely we give the baby 5 mgm. of Vitamin K, intramuscularly, almost immediately after the umbilical cord is cut. Furthermore, each mother-to-be is given 10 mgm. of Vitamin K during labor. Other medications can be given to her as indicated for the benefit of the baby.

The essayist has done well to mention the very important group of babies born of patients having complicated labors terminated by operative procedures after much sedation, and I agree with his statement regarding the unfortunate situation of these individuals in later life.

I had hoped that this presentation would not include the word "simple". I wish to suggest that

it be changed to a "rational plan." In this connection may I deplore the fact that too many lay publications have ballyhooed *simple, safe, painless* childbirth methods as Dr. Dyer says, "There is really no such thing."

Each case must be individualized. There is no substitute for obstetrical judgment.

Saddle block for the delivery of premature infants is indeed fine. It permits the administration of oxygen to the baby by way of the mother as indicated. Likewise we advocate its judicious use in certain cases of fetal distress or in which the fetus is apt to become distressed. Its use for the last two or three hours of many labors is also found to be highly satisfactory; however, one must be mindful of the fact that some of the headaches following the saddle technic are difficult to control by the intravenous administration of 50 per cent dextrose, pressure to the abdomen, or even opiates. And furthermore, it must be expected that some of the patients will hold the opinion that their postpartum backache is due to the spinal puncture. There again one must individualize and rationalize.

Regarding spinal analgesia for cesareans, may I state that I have used such since my days as a resident at Charity Hospital. Obviously one must not allow the analgesic agent to ascend to a dangerous level. Ideally a well trained anesthetist performs the intrathecal injection. If it is necessary for the operator to administer the drug in a small hospital, he must carefully and repeatedly check the level of anesthesia, the blood pressure, the pulse rate, and see that the infusion is progressing properly. That means that he must wait until the analgesic agent is fixed and be sure that the patient is in good condition before making the skin incision. Certainly a competent anesthetist must be at the head of the table constantly to administer oxygen as necessary, and to observe the blood pressure readings, pulse and respiratory rates, and color of the patient. One should promptly clear the upper respiratory passages of babies delivered by spinal sections to prevent aspiration by them of amniotic fluid and, in some cases, blood, inasmuch as they usually take a breath as soon or shortly after the face is exposed. By prompt action one can eliminate the necessity for passing a catheter into the trachea. It is agreed that cyclopropane is a fine anesthetic agent when properly administered. Because of its great potency, it requires a skillful anesthetist well versed in its use.

I have very much enjoyed Dr. Dyer's paper and I repeat for emphasis his opening sentence. "The ideal agent to employ routinely in order to obtain analgesia and/or anesthesia in obstetrics has yet to be found."

Thomas B. Sellers, M. D. (New Orleans): I should like to endorse the comments which Dr. Beacham has just given to Dr. Dyer's paper.

First, I should like to ask of Dr. Dyer what treatment he administers for, or how does he control, post-saddleblock headaches?

I wish to mention and emphasize the subject of fluids, which Dr. Dyer most ably discussed. At times, it is very difficult to maintain fluid balance in cases of prolonged labor, especially if the patient does not have the services of a special nurse. I think it should be routine to administer intravenous glucose to patients in labor over twelve hours.

The patient should be told to eat and drink very little after she notices early labor pains. Though these nagging pains often last for hours and we find the patient dehydrated, still, if much food is consumed, we find it remains in the stomach, a hazard for the anesthetist. It was my misfortune to lose a patient from aspiration pneumonia. I also want to endorse the precautionary measures which Dr. Dyer mentioned concerning the anesthetist, but I should like to emphasize this point: The average anesthetic accident is rare enough so that it is very difficult to get cooperation in having an adequate stomach tube in the delivery room. In the hospital in which I work, the tube is often lost when needed .

I also want to emphasize and reiterate the use of oxygen during anesthesia, administering as high a concentration as possible before the baby arrives.

Dr. Isadore Dyer (In conclusion): I think if any one asks Dr. Adriani, who we think is the father of saddle block anesthesia in obstetrics, the treatment for post spinal headaches, he would reply that he had none. We may go for months without seeing any postdelivery headaches, then all of a sudden we may have two or three on the wards. At one time we thought that drug sensitivity played some part. I believe that one thing that will prevent postspinal headaches is not to ambulate your patient too soon. Usually if they stay prone without a pillow for twelve hours after delivery, and then are slowly introduced to ambulation, postdelivery headaches are averted. If they have the slightest headache we keep them down flat for a couple of more days. In any case I have seen few headaches persist for more than two to five days.

As for the remark about food, we are impressed with the fact that many multiparous women who progress rather rapidly, often eat before they realize they are in active labor. Should labor begin shortly after a full meal a serious complication might develop when inhalation anesthesia is employed. I think that you should caution your patients not to eat if they think they are in labor.

Fluids by mouth, as a rule, will be digested rather well and certainly fluids aspirated are less dangerous to the patient than solid food aspirated.

I agree with Dr. Sellers that you should watch dehydration during the warm months. Obstetric

procedures on an emergency basis should be preceded by gastric lavage if the patient has recently eaten.

I want to thank Dr. Beacham for his good remarks, and I shall change my wording from a "simple" plan to a "rational" plan.

—o—

THE MANAGEMENT OF ACNE IN GENERAL PRACTICE

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Acne vulgaris is a chronic inflammatory disease of the pilosebaceous system manifested as comedones, papules, pustules, cysts, and nodules. The comedone or blackhead is the primary lesion being derived from the fatty plug at the orifice of the sebaceous gland. The site of predilection is the face although the back, chest and arms are commonly involved. Seborrhea as a rule is found associated with acne.

Acne vulgaris is slighuly more common in the male than in the female,[1] occurring most frequently during adolescence, appearing at a younger age in the female than in the male, with incidence of occurrence at its peak at 15 years. However, there is no age group exempt as acne is found from infancy to old age. We were unable to obtain any racial statistics; however it has been our observation that acne is comparatively uncommon in the Negro, and in the white race those with darker skin suffer less frequently than the blond type:

TYPES

There are many different types of acne vulgaris clinically, the most common types being:

1. The *comedo* type, consisting chiefly of blackheads and an oily skin.

2. *Acne papulosa*, small papules associated with numerous large comedones that have become inflamed causing a diffuse redness of the involved skin.

3. *Acne pustulosa*, composed of small superficial pustules and few comedones.

4. *Acne indurata*, deep seated indurated lesions as the result of coccigenic infection of papules. These may be destructive causing marked scarring.

5. *Cystic acne*, subsequent to the inflammatory type of acne leaving an oily seborrhea and inspissated comedones and cyst. The contents of the cyst are usually a blood tinged gelatinous fluid.

6. *Acne conglobata*, uncommon type, in which the comedones, papules, and pustules are associated with large abscesses, cysts, and discharging sinuses, which heal slowly and leave pronounced scarring and often keloid formation.

7. *Acne keloid.* Lesions are located on the back of the neck caused by the presence of numerous large or multiple comedones burrowing under the skin and becoming infected, leading to pustules and residual keloid formation. This type is seen commonly in the male especially of the Negro race.

ETIOLOGY

The etiology of acne vulgaris is unknown, but apparently several conditions must exist before the reaction in the pilosebaceous system manifests itself as one of the clinical types of acne.

Stokes and Sternberg[2] in reviewing 921 cases of acne from an etiological standpoint found several factors of importance:

1. The *familial factor.* They found that the probability of the appearance of acne in a patient was some 26 times greater if there had been previous cases in the family. This they felt was due to a greater susceptibility to pyogenic infection.

2. *Age, endocrine factor.* Consistently emphasized though it has been because of the association of acne with the puberal years, the endocrine factor in the causation of acne when brought down to present biochemical knowledge is disappointing. Considerable impetus was given to the study of endocrine balance in acne by Rosenthal and Kurzrok,[3] who found no gonadotropic substance in the urine of 34 young women with acne. The estrus-inducing hormone was normal in 6 cases, present in traces in 1

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case, and completely absent in 27 cases. The favorable therapeutic application of these findings as reported by Lawrence and Feigenbaum,[4] Rosenthal and Newstaedter,[5] and Lawrence[6] by prolonged series of injections of gonadotropic substances (antuitrin-S) was not confirmed by later reports of McCarthy and Hunter[7] and Williams and Nomland[8], using the same method of treatment.

3. *Hyperactivity of the sebaceous glands.* The activity of the sebaceous glands as an underlying factor in the acne complex is illuminated by several considerations, including first the evidence for a thalamic center of fat secretion. Experimentally it has been shown in rabbits, in which a lesion of the midbrain has been produced, that excretion of fat by the skin increases as much as 200 per cent. This is confirmed to some extent clinically by the high grade seborrhea commonly found in young women during an emotional upset or a depressive state. The activity of the sebaceous glands is also dependent on the carbohydrate intake, increasing greatly when the diet is high in carbohydrates.

4. *The scalp tie-up.* It seems that in acne with seborrhea of the scalp that the scalp acts as a focus of infection and as a sensitizer for the face and back similar to that seen in seborrheic eczema.

5. *The allergic element.* The apparent specific allergic reactions with decided flare-up of the acne after ingestion of chocolate or milk or other specific foods, and quiescence when these foods are eliminated from the diet. The infection-allergy phase of the acne complex has been confirmed by the flare-up or relapse of the acne with intercurrent infections in other systems as in gastrointestinal upsets, and upper respiratory infections.

6. *Water balance factor.* There is considerable experimental and clinical evidence that hydration of tissue is a direct predisposing factor to infection particularly to pyogenic infections. The hydration of tissue may be unfavorably influenced by a high carbohydrate intake, and alkaline ash diet, and by the action of ovarian and testicular hormones as observed clinically in association with the cycle of ovulation and menstruation in women.

7. *Contact inoculation factor.* Picking and squeezing of comedones and pustules with inoculation of adjacent normal sebaceous glands.

8. The *rosacea background.* The acne frequently observed in those individuals who blush from the slightest embarrassment, the lesions having a distribution confined to the flush areas of the face.

9. *Fatigue and exhaustion.* The frequent observation of the exacerbation of acne when the patient is under strain, as in college or high school students during examinations or periods of exhaustive social activity.

10. *Vitamin, anemia, and constipation.* The pustular secondary involvement of the follicular keratoderma observed in avitaminoses in man suggest the desirability of liberal doses of vitamin A in treatment of the acne patient. The work of MacKay and Clausen[10] seems to indicate a definite increase in the resistance of the skin of infants receiving adequate amounts of vitamin A.

The vitamin B complex contains a number of elements for the management of acne patients. Vitamin B_1 has a beneficial effect on the motility of the gastrointesinal tract and should be used alone or as the B complex for the management of constipation in acne patients. The B complex also contains an achlorhydria preventive fraction which helps the utilization of the hematinic agents, as hydrochloric acid is essential to the proper and full utilization of these agents.

11. The *psychoneurogenic* factor. May affect the acne patient by emotional effects on the thalamic centers controlling fat secretion; the ill effects of vasomotor instability particularly of the face and neck; the depressive effects of emotion on the secretion of hydrochloric acid, and the action of emotion on the endocrine balance.

12. *Industrial* factor. Workers in industries, where prolonged contact with greases, oils, tars, and similar hydrocarbons is nec-

essary, frequently develop acne or a preexisting acne may be aggravated.

13. *Iodide and bromide* factor. The ingestion of these halogens apparently aggravates acne and may produce acniform eruptions.

PROGNOSIS AND SEQUELAE

The prognosis and sequelae of acne vulgaris depends on the type of acne, the cooperation received from the patient in treatment and the time that treatment is instituted. However, we feel that it is generally agreed that one may expect good results in approximately 65 per cent of the cases, an improvement in 20 per cent and and about 15 per cent failures. The final results, in the most favorable response, will leave some superficial negligible scarring in the mild or moderate cases, to deep disfiguring scars and keloid formation in the more severe types of acne.

TREATMENT

In acne vulgaris, as in any other disease of really unknown or multifactored etiology, treatment cannot be classified as specific and nonspecific or supportive. Therefore, we shall not impose upon you "our" treatment and close the discussion of other therapeutic phases. Instead we shall discuss all suggested phases of therapy endeavoring to bring them up-to-date and help evaluate them in our opinion.

Therapeutic agents and classes may be grouped as follows:

A. Psychological:

It is quite satisfying to note the change in many persons' personalities paralleling the improvement of their acne. There arises also the psychosomatic question. Mitchel Heggs[11] states acne often produces an inferiority complex, later an anxiety state. Some writers state the reverse is true.

At any rate the patient should be treated from a psychological viewpoint. Untold psychic trauma has been dealt many patients by telling them not to worry, their skin will clear up with age. These patients should be reassured—to place their minds at rest should there be a psychic etiologic factor, and secondly, to assure their cooperation for the long treatment period ahead.

B. Constitutional:

The general patient should of course receive attention, not just the presenting complaint. Rest, anemia, foci of infection, constipation, and intestinal parasites should be considered and investigated. Some authors stress some, minimize others. Osborne[12] minimizes the role of constipation, Miller[13] suggests control of this complaint by diet and exercise. Habits of traumatizing the face with the nails and fingers should be strongly discouraged. It is a fairly common observation to see the right side of the face markedly more severe than the left merely because most people being right handed, that side of the face is more "accessible."

C. Local Therapy:

1. Topical: This undoubtedly is the most frequently used and abused form of therapy. Strong or irritating preparations are neither necessary nor beneficial. Since the overwhelming majority of patients suffer from excess oiliness, a removal of these oils is indicated. Klauder[13] recommends soap, water and the alcohols. Swartz and Blank[14] report the use of a detergent composed of 25 per cent sulfated oil (emulsifying agent), 25 per cent mineral oil, and 50 per cent water, as a cleansing agent used in 400 patients with good clinical results. They recommend its use three times daily on an unmoistened skin and removal with warm water.

Soap and water seem adequate in most cases as a cleansing agent. However, it must be borne in mind that many skins are chapped from overdrying by soaps, in which case its use must be decreased or some milder soaps used. Strong or medicated soaps are not advisable.

Keratolytic ointments and sulfur ointments or their combinations are still in wide use and are often beneficial if judiciously used. New preparations combining these medicaments have appeared in wetting agents and cosmetic bases. It seems questionable how much the efficacy of these drugs has been increased thereby.

2. Surgical: Some surgical procedures be-

come indicated in almost all cases. We believe they should be minimized as much as possible. MacKee[13] recommends no surgical procedures, while Klauder, Michelson, Sutton and Wile[13] advise incision of pustules and extraction of comedones. Warren[15] has suggested aspiration of cysts and possibly replacing the contents with penicillin.

Incision of lesions with a cataract knife is adequate in most cases and minimizes trauma and scarring. Comedone extractors are likewise helpful.

D. Physical Therapy:

1. X-ray: We believe this to be the most efficient single agent in our hands today. Its use should be limited to one thoroughly familiar with it. X-ray therapy in full tolerance doses is not 100 per cent effectual and failures are not uncommon. We further advocate its use be withheld except in very severe cases until the age of 15 or 16 has been reached.

2. Ultraviolet Light: Unlike x-ray, this form of physical agent has no depressive effects upon overactive oil glands. Its effect is usually temporary and its uses seem to be (1) merely as another peeling agent and one can cause peeling with any speed desired by varying the dosage; and (2) bacteriocidal, thereby reducing the bacterial flora of the skin.

3. Cryotherapy: Is not widely used but mentioned for thoroughness. A slush is made of carbon dioxide snow, sulfur, and acetone, and applied with a tongue depressor. Marshall[16] advocates it in failures from other treatment, dry skins, and in indurated areas.

E. Hormonal:

Liver, thyroid, pituitary and ovarian substances have all been used with great variance of opinion. Nyvell[17] states they are the treatment of choice. MacKee believes they should be used only if indicated by symptoms other than acne, Sutton advocates thyroid in all cases and paratharome in extremely deep seated types. Wile uses them only for menstrual disorders while Michelson and Miller believe them to be of no value. Osborn[12] agrees in the routine use of thyroid and sees no necessity for a routine basal metabolic rate.

An improvement of 82 per cent is reported with pregnant mares serum in a group of cases with menstrual disturbances by Birnburg and Rein.[22]

We believe the use of hormones should be restricted to an attempt to regulate menstruation or other glandular disturbances. The absence of such an indicator as the menses in the male leads to their more universal use in that sex.

F. Diet and Vitamins:

Dietary restrictions are fairly uniform and eliminate or restrict chocolate, iodized salt, fatty[18, 19] greasy, and highly seasoned, sweet, and sea foods.

Straumfjord,[20] stressing that acne is due to hyperkeratosis of the pilosebaceous follicle, reports 79 per cent of 100 patients free or nearly so on 100,000 units of vitamin A, daily, for six months or more.

Stokes and Sternberg[2] recommend a high vitamin therapy stressing vitamin A for follicular keratoderma and vitamin B, for constipation.

Pyridoxine has been advocated but any explanation of its role is at present speculative. Jolliffe[21] and his co-workers reported encouraging results using on an average of 25-50 mgm. daily initially, the dosage being decreased or increased as indicated clinically.

C. Vaccines, Antibiotics, and Drugs:

Staphylococcic vaccines, toxoids, mixed vaccines and autogenous vaccines have been used and various authors are not in agreement on their benefit. We believe them rational to build up a resistance to secondary invaders but of no value as a primary therapeutic agent.

The use of the various antibiotics and chemotherapeutic agents is attacking the problem from the same point of view as in the use of vaccine therapy only their effects are even more temporary and secondary than those of vaccine therapy.

H. Cosmetics:

The question of their use over a skin affected with acne always presents itself in

the handling of female patients. The use of the type of cosmetics that produce a cake-like mask and mechanical follicular plugging, even though temporary, seems contra-indicated. Otherwise, cosmetics should be allowed provided there is no allergic element. Female patients are much more likely to cooperate if cosmetics are not ruled against.

I. Seborrhea:

The treatment of this condition will not be dealt with here. However, because of its frequent occurrence in acne patients, its treatment should be incorporated with treatment of acne.

CONCLUSION

It is not wise to form an ironclad "routine" of therapy but some nucleus of therapy should be arrived at by the clinician upon which to build the therapy. This nucleus of therapy will naturally depend (1) upon one's ideas of etiology, and (2) upon one's experiences with the various medicaments. The addition to or subtraction from this nucleus of therapy should depend upon the presenting clinical features.

Every case of acne cannot be treated with all suggested forms of therapy and the various clinical features of each case must determine which form or groups of forms of therapy will be followed.

REFERENCES

1. Block, B.: Metabolism, endocrine glands and skin diseases with special reference to acne vulgaris and xanthoma, Brit. J. Derm. 43:61 (Feb.) 1941.

2. Stokes, John H. and Sternberg, T. H.: A factor analysis of the acne complex with therapeutic comment, Arch. Dermat. & Syph. 40:345, 1939.

3. Rosenthal, J. and Kurzrok, R.: Excretion of estrin in acne, Proc. Soc. Exper. Biol. & Med. 30:1150 (May) 1933.

4. Lawrence, C. H. and Feigenbaum, J.: Treatment of acne vulgaris with pregnancy urine extract, New England J. Med. 212:1213 (June 27) 1935.

5. Rosenthal, T. and Newstaedter, T.: Estrogenic substance in blood of patients with acne, Arch. Dermat. & Syph. 32:560 (October) 1935.

6. Lawrence, C. H.: Anterior pituitary-like hormone: Clinical study of its effects in acne vulgaris, J. A. M .A. 106:983 (March 21) 1936.

7. McCarthy, L. and Hunter, O. B.: Failure of therapy with glandular preparations in acne vulgaris, Arch. Dermat & Syph. 35:211 (Feb.) 1937.

8. Williams, G. E. and Nomland, R.: Gonadotropic substance in treatment of acne, J. A. M. A., 109:564 (August 21) 1937.

9. MacKay, H. M.: Vitamin A deficiency in children, Arch. Dis. Childhood 9:65 (April) 1934.

10. Clausen, S. W.: Nutrition and infection, J. A. M. A. 104:793 (March 9) 1935.

11. Mitchell-Higgs, G. B.: The Practitioner, 157:379, (Nov.) 1946.

12. Osborne, Earl D.: Treatment of acne, Post Grad. Med. 1:16 (Jan. 1947).

13. Miller, Klauder *et als.*: Symposium on the Practical Management of acne vulgaris. J. Invest. Derm. 3:143 (April) 1940.

14. Swartz, J. H. and Blank, I. H.: The use of sulfated oil as a skin cleanser, in the management of acne vulgaris, J. A. M. A. 125:30 (May 6) 1944.

15. Warren, C. M.: The modern treatment of acne, Med. Press 216:115 (August) 1946.

16. Marshall, Wallace: Old and modern treatment for acne vulgaris, Tri-State M. J. 14:2603, 1942.

17. Nyvall, H. O.: Treatment of adolescent acne with endocrines; Urol & Cutan. Rev. 47:685, 1943.

18. Shelmire, B.: Acne from iodized salt, J. A. M. A. 90:1869 (June 9) 1928.

19. Wise, F. and Sulzberger, M.: Yearbook of Derma. & Syph. 1933 P. 10.

20. Straumfjord, J. V.: Vitamin A., its effect on acne. Northwest Med. 42:219 1943.

21. Jolliffe, N., Rosenblum, L. A. and Sawhill, J.: The effects of pyridoxine (vitamin B6) on persistent adolescent acne, J. Invest. Derm. 5:143 (June) 1942.

22. Bimberg, C. H. and Rein, C. R.: Treatment of acne vulgaris, J. Clin. Endoc., 4:65 1944.

DISCUSSION

Dr. M. T. Van Studdiford (New Orleans): It is a shame that two such excellent papers should have fallen upon the ears just of dermatoligists and not upon the ears of general practitioners, surgeons, gynecologists, and obstetricians, all of whom treat acne.

I can add only praise to the gentlemen for their thorough work-up of their papers. Dr. Bradley* very mildly claimed what results he got from para-aminobenzoic acid. He very graciously did not claim too much credit but left a loophole. He is perfectly justified. I saw Dr. Bradley's patient, and I will say para-aminobenzoic acid might be a drug for trial in cases of lupus erythematosus. From nearly a morbid state it brought his patient to a state of comparative wellbeing, and although not well, he was walking around in perfect comfort.

Acne is such a bugbear to dermatologists because of the superstitions of the public; the superstitions that x-ray makes scars, and that the doctors make the patient worse, are so instilled in the people that it really raises your blood pressure when you hear the weird expressions from patients, their friends, and some poorly informed of the profession.

People go to their doctor because they have acne, not because they want it produced. But they would like to have the public believe that the scars and pustules they have on their face were put on there by treatment, and their mothers would like to place the blame not on the changes of

*Subacute disseminated lupus erythematosus by David F. Bradley, M. D., New Orleans M. & S. J. 102:83, (August) 1949.

adolescence, but on the dermatologist, or on the x-ray treatment that is subsequently taken.

I do not think sugar has any effect at all on acne, unless the person is a potential diabetic. So I get my blood pressure up when patients tell me they have to be dieted and they do not want to be x-rayed. If I feel that x-rays are justified, they are either treated by x-rays or by someone else. I don't believe you can take a superstition and work it into some good routine manner of treatment.

Now it is known that if a person squeezes blackheads or pustules without removing the plug, or without making an orifice to let the material be expelled, they are going to force the bacteria into the surrounding tissues and make it worse than if it were left alone. I believe in opening the pustules and expelling the caseous mass that must otherwise be absorbed, and when it is absorbed you have an erythema and the pathological stimulation makes the acne worse.

I think we should, as dermatologists, say that x-ray does not cause scars, and that the scars are the result of abscesses, and if the abscesses are not allowed to form they do not get as large a scar as if the abscesses are allowed to spread into all dimensions. We must impress upon people that they inherit the skin that covers them. It is not wished upon them by nature after they have been born, but it was wished upon them while they were in the womb, and they are going to carry that skin through life. If they have a chronic, 15 per cent failure type of acne, impress that on these people so that they will not blame the dermatologist, but blame their parents or forebears, whoever gave them their type of skin.

It is not a very easy thing to impress upon a person, but after a while they look at themselves in the mirror, and look at home to see whom they take after, and look at the scars on the old lady and on the grandmother, and begin to figure that the old lady and the grandmother were lying a little when they said they didn't do this or didn't do that.

Dr. J. K. Howles (New Orleans): A few points on acne still stare us in the face. The thing that puzzles me is why do we have more acne—and we definitely do; it is not imagination—in the younger generation than we had previously? I have thought of it from several angles. Certainly from the glandular standpoint, there should not be any difference from previously. I have considered the partially sedentary mode of existence; today people get in the automobile or bus, where formerly they walked. While they do have exercise in school, it is limited to gymnasium work which is a poor excuse for routine exercise, and they get that once a week. Another point is diet: I believe I agree with Van Studdiford that it is less important in the general run of acnes.

There is a group, in my opinion, that acts entirely differently from the general rule of acnes, as the acne is prone to persist after passing the age of puberty into young adult life and even into late adult life. I have no solution to it. I think the sedentary mode of existence and the fact that we have more chocolate bars and sundaes—that's possible.

The only thing that impresses me is the fact that people come and tell you about x-ray scarring the face. I agree that you should educate people to the fact that it is the pathology of acne that accounts for the scarring and the lesions. Now, how x-ray can produce a pinpoint scar is beyond me. I have never had anyone explain how or why x-ray could produce a pinpoint scarring.

Another thing is having the x-ray treatments done at the local hospital instead of the private laboratory. I definitely object to using the hospital as your laboratory; I think the private clinician should have a chance at that before we support the institutions. The thing that amuses me (and I am sure you have all had the same experience) is that when we go to academy meetings, which to us is like going to Mecca, we have a symposium on acne, and someone has the job wished on him to present the subject. We have the symposium on acne and it usually boils down to the same remarks: X-ray is the best single treatment; opinion on diet varies with different people; the hormone factor, about the same as described by Dr. Jolly. X-ray is the best single treatment.

The one thing I want to add is concerning the misconceptions and erroneous idea that acne gets worse during pregnancy. In my opinion that is not so. Many cases clear up during pregnancy and when the menstrual cycle starts again, the acne is back again.

Dr. C. B. Erickson (Shreveport): I have no original views about acne. I am in accord with the views of the essayists and pretty much in accord with what the two previous discussors have said. I will say, however, that there isn't anything that I do and have done in thir tyyears practice of dermatology that has given me quite the satisfaction that treating acne has. Granting it is not perfect, but if you can help those cases of depression and inferiority complexes that these young folks have, and give them a little brighter outlook on life, you have really done something that is worthwhile.

I don't think that any dermatologist would question the fact, as stated, that X-ray is the best single remedy we have for acne. I want to make a little comment about what Dr. Van Studdiford brought out about scarring and blaming the treatment for it. When that situation prevails, it's somebody's fault that the idea has been allowed to get in the patient's and their families' mind that acne is what produces the scarring and not the X-ray. I don't have that trouble at all any more. It takes only a few minutes to explain and convince the patient and the patient's parents that

scarring is due to the disease and not the treatment.

I don't see eye to eye with everybody here about the question of diet. I'm rather inclined to think it plays a rather prominent part. Nor do I subscribe to the idea that constipation has nothing to do with it; I don't think you can get best results without proper elmination.

Thyroid when indicated is a wonderful help, but it should not be given routinely. The same is true for ovarion preparations; only when really necessary. There is no specific for acne but I think if one takes the trouble and time to combine what we know, we can do a good job in this disease.

Dr. J. D. Youman (Shreveport): I have nothing to add to what has been said. I would like to stress the removal of blackheads as Dr. Van Studdiford already has. I think by carefully doing this, you can minimize the scarring, and progress is faster. Care must be taken not to bruise the lesion.

Vitamin A was mentioned. I have not had any luck with Vitamin A, using 100,000 to 150,000 units a day for three to six months. In the first place, you have to wait three to six months before you can tell whether the vitamin A is helping. During that time, it is hard to tell if it is the vitamin A or the other things that you have done which have caused improvement.

Recently, at a meeting in Chicago, Dr. Stoesser was talking about infantile eczema. He said he was getting results with a new synthetic vitamin A preparation. It is not yet on the market.

A CASE OF BILATERAL SPONTANEOUS PNEUMOTHORAX*

JACOB FAUST, M. D.†

BATON ROUGE

Spontaneous pneumothorax in the apparently healthy has been a well recognized disease entity at least since Kjaergoard's monograph[5] in 1932. It is not an unusual disease. There were 873 cases in the U. S. Army in 1943.[13] Schneider and Reissman[11] estimated that about 1 in 500 of the inductees in New York gave a past history of this condition. Perry[9] was able to collect 67 cases from the London Hospital in fourteen years, from an annual admission of about 10,000. At Charity Hospital, in the past five years, from an annual admission of about 50,000, there have been 12 cases.

Spontaneous pneumothorax is a disease of sudden onset, most common in young males, not necessarily associated with severe muscular activity. It is usually initiated by a dull pain in the chest which increases and becomes stabbing in character. It is relieved somewhat by leaning forward, and is associated with tachycardia and usually some degree of dyspnea. In rare instances it is associated with no symptoms whatsoever, being picked up on a routine chest plate. The usual physical and x-ray signs of pneumothorax are present and there are generally none of the changes in the white count, sedimentation rate, and temperature, usually associated with infection. There may be a little fluid in the costophrenic angle. The condition is benign, the lung tending to re-expand spontaneously within a few weeks in the majority of cases, though there are some on record in which a bronchopleural fistula and pulmonary collapse have persisted after twenty years. However, there is a tendency to recurrence, about 20 per cent of the patients having two or more attacks, usually on the same but often on opposite sides. Tuberculosis is no more common in these patients than in the general population.

The majority of these cases have been demonstrated to be due to one way valve vesicles, a portion of the lung being progressively distended by intake without egress of air until the limit of elasticity of the tissue is exceeded and rupture results. Among the causes of these vesicles may be mentioned scar tissue, localized emphysema, partial bronchial obliteration and congenital blebs of the pleura. These latter can often be demonstrated the fluoroscopy or at operation. The term spontaneous pneumothorax has over a period of time come to be applied largely to those cases who show no historical or x-ray evidence of associated disease, and should be differentiated from a much larger group with spontaneous collapse due to tuberculosis, asthma and emphysema, cystic disease of the lung, empyema, pneumokoniosis, postoperative dissec-

*Presented at meeting of the Orleans Parish Medical Society, February 14, 1949.

†Formerly Resident in Medicine, Charity Hospital of Louisiana, New Orleans.

tion of air through fascial planes, and the fairly common pneumothoraces in newborns which are probably related to over inflation of portions of the expanding lung. The patients, except for their tendency toward repeated collapse, are usually healthy, and death, when it occurs, is due to one of three rare complications: hemopneumothorax, tension pneumothorax, and simultaneous, bilateral collapse. This latter complication is the subject of this paper.

CASE HISTORY

The patient a healthy, hypersthenic, 23 year old, white male, a semiprofessional trombone player, was first seen on December 19, 1948. He stated that he had been playing touch football and had run out to catch a pass. On returning to the line of scrimmage, he first noted a dull pain in the right upper chest. There was no particular dyspnea, but he sat on the ground, expecting the pain to pass. Instead, it became progressively more severe and stabbing in character. It finally became so severe that an ambulance was called and he was brought to the hospital. There increased resonance over the right lung field was noted and absent breath sounds. An x-ray showed approximately 60 per cent collapse of the right lung (Fig. 1).

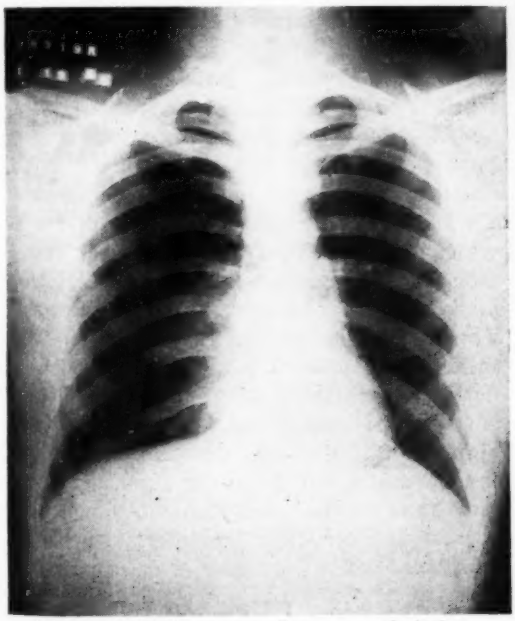

Figure 1—Right pneumothorax on admission.

(This x-ray is misdated and should be December 19, rather than January 19.) He was admitted to the ward where his temperature was normal; pulse rate was 100; white count 15,000 with 76 per cent polys, and sedimentation rate 9. He was placed on bed rest, codeine for the control of cough, and assured that this was a benign condition from which he could be expected to recover quickly. About 4:30, on the morning of December 22, he began complaining of severe pain in the left chest, and fairly marked dyspnea was noted. His respirations increased to 40 per minute. He sat up in bed, gasping for breath, and a slight amount of cyanosis was noted. A water trap was set up on the left side with some relief. However, during the later course of the morning, his dyspnea continued to increase, he became much more apprehensive, and x-ray taken at this time showed approximately 70 per cent collapse on the right and 50 per cent collapse on the left (Fig. 2). Approximately 2,000 cc. of air were removed from the right pleural cavity and a water trap placed on this side as well. Marked dyspnea continued throughout the day in spite of oxygen and the use of the water traps. Toward evening the patient was febrile, with a temperature of 101° F., and drainage of a purulent fluid from the left pleural cavity. Patient had been started prophylactically on penicillin at the time of placing the second water trap, however, and cultures of the purulent fluid at this time and subsequently failed to demonstrate any organisms. The needles tended to become blocked by the exudate, and, in view of the continuing dyspnea, the Surgery Department was consulted. Soft rubber catheters were placed in both pleural cavities through trocar wounds; these

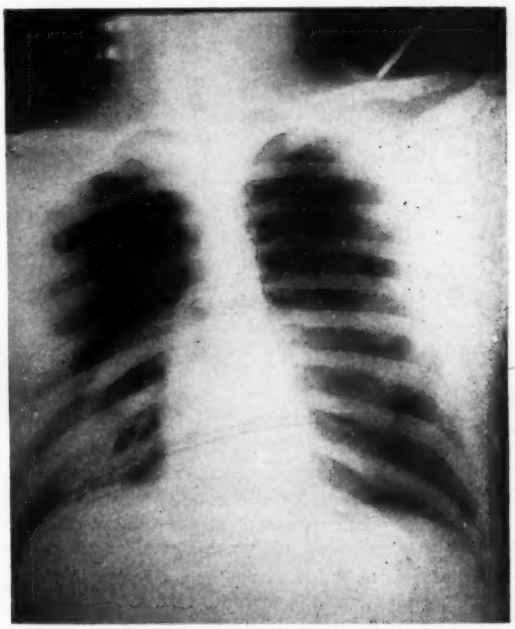

Figure 2—Bilateral pneumothorax approximately 8 hours after onset.

were connected to water traps and the result was a rather considerable improvement in the degree of dyspnea. Patient's course from then on was essentially uneventful. He continued spiked temperature between 101 and 102° F. until approximately December 29, and then over the next week it gradually declined to normal. By December 29, dyspnea had improved sufficiently so that it was possible to remove both catheters. X-ray at this time showed almost complete re-expansion o fthe right lung and expansion to about the level of the clavicle on the left (Fig. 3). There was a persist-

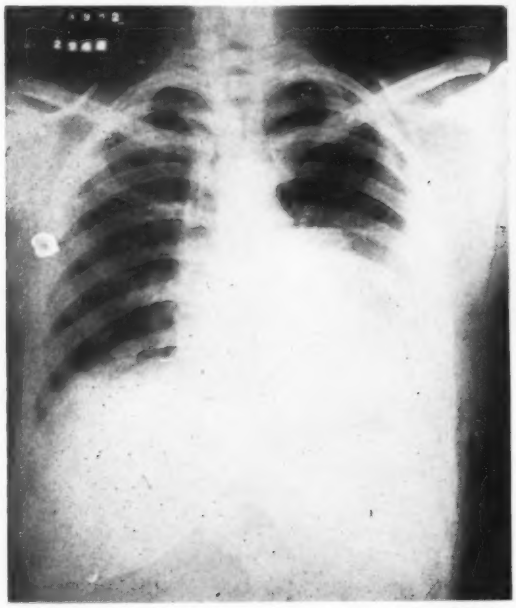

Figure 3—Almost complete re-expansion bilaterally shortly before discharge December 29, 1948.

ent haziness at the left base, probably due to pleurisy. Penicilling was discontinued on January 7, and the patient discharged on January 13, comfortable. A subsequent x-ray on February 4, 1949, showed complete re-expansion of both lungs (Fig. 4).

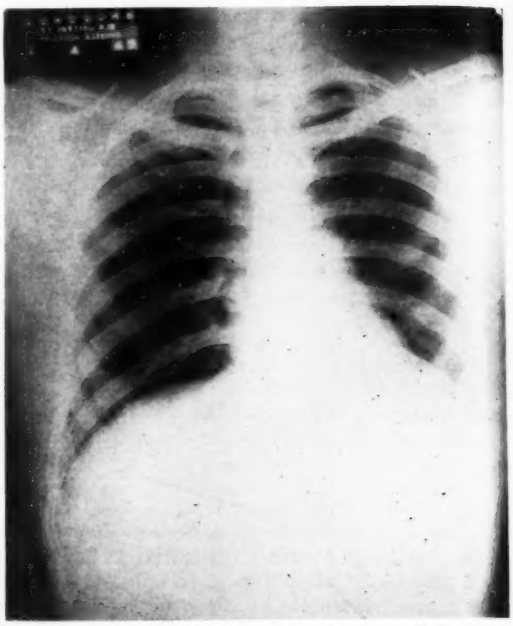

Figure 4—Apparently normal x-ray February 4, 1949.

Various causes have been suggested for these bilateral collapses. Dissection of air through the mediastinum or abnormal mediastinal passages have not been demonstrated in the autopsy cases; nor have any of the authors commented on crunching respiratory sounds in the sternal area. The best explanation seems to be that the same pathology exists in both lungs and that the hypernea, overdistention, and mediastinal shift induced by the first collapse helped bring on the opposite one. Oechsli and Miles[8] reviewed the literature in 1934, and found 17 cases of this type, of which 4 were fatal. They apparently missed the case of Markson and Johnson,[7] as it does not appear in their index. Hasney and Baum[3] found 2 cases of Rossel, with 1 death, and added 1 of their own. Since then there have been case reports by Priest[10] with death and a normal autopsy, Werner and Thearle,[13] with death and emphysema which was not apparent on x-ray at postmortem, Baum,[1] Castex and Mazzei,[2] King and Benson,[4], with death, and Komrower.[6] These, with the present case bring the total in the literature to at least 28 with 8 deaths, a mortality of 28 per cent. The supplemental index lists 6 cases in the foreign literature which was unavailable to me, so that I am unable to decide if they meet the criteria heretofore laid down.

Various methods of treatment have been used—needles with water traps, one way rubber valves, repeated aspiration, and catheters with varying types of suction. No one individual has sufficient experience to evaluate various modes of therapy, but it would seem necessary to re-expand as rapidly as possible at least one of

the lungs, preferably the one first collapsed as its rupture is more likely to have sealed off. This may at times, with a large fistula, require the removal of considerable quantities of air, and machine suction through a large catheter may be necessary.

SUMMARY

1. A case of bilateral spontaneous idiopathic pneumothorax in a healthy young male is presented with recovery following water trap suction through rubber catheters in both pleural spaces.

2. This is the twenty-eighth such case reported in the literature.

REFERENCES

1. Baum, F.: Rare cases of bilateral idiopathic spontaneous pneumothorax in apparently healthy individuals. Bull. Am. Acad. Tuberc. Physicians 5:19, 1941.

2. Castex, M. R., and Mazzei, E. S.: El neumotorax espintaneo bilateral simultaneo. Prensa. Med. Argent. 21:2187, 1940.

3. Hasney, F. A., and Baum, F.: Idiopathic spontaneous pneumothorax in apparently healthy individuals. Radiology 28:47, 1937.

4. King, A. C., and Benson, W.: Bilateral spontaneous pneumothorax. J. A. M. A. 125:782, 1944.

5. Kjaergard, N.: Spontaneous pneumothorax in the apparently healthy. Acta. Med. Scandinav. Supp. 43, p. 159, 1932.

6. Komrower, G. M.: A case of bilateral simultaneous spontaneous pneumothorax. Brit. M. J. 1:488, 1947.

7. Markson, D. E., and Johnson, W.: Simultaneous, bilateral spontaneous pneumothorax. J. A. M. A. 102:826, 1934.

8. Oechsli, W. R., and Miles, S. H.: Simultaneous, bilateral spontaneous pneumothorax. Am. Rev. Tuberc. 30:239, 1934.

9. Perry, K. M. A.: On spontaneous pneumothorax. Quart. J. Med. 8:1, 1939.

10. Priest, R.: Bilateral spontaneous pneumothorax. Brit. M. J. 2:321, 1937.

11. Rossel, G.: Quoted in (3). Rev. med. de la Suisse Rom. 55:849, 1935.

12. Schneider, L., and Reissman, I. I.: Idiopathic spontaneous pneumothorax. Radiology 44:485, 1945.

13. Werner, W. I., and Thearle, W. H.: Spontaneous bilateral pneumothorax. Am. Rev. Tuberc. 46:105, 1942.

14. News & Comment, U. S. Army Med. Bull. 82:29, 1944.

SUPPLEMENTARY REFERENCES

1. Castello-Branco, J. M.: Pneumothorax espontaneo bilateral simultaneo. Pleuroscopia e pleurodex bilateralis. Clin. tisial. 2:213, 1947.

2. Passalacqua, H.: Pleurodisis with simultaneous bilateral spontaneous pneumothorax, 29:337, 1942.

3. Poli, E.: Simultaneous bilateral spontaneous pneumothorax. Riv. Clin. Med. 40:190, 1939.

4. Quero-Morente, M.: Simultaneous bilateral spontaneous pneumothorax. Rev. clin. espan. 1:57, 1940.

5. Sattler: Rasche Heilung lines bilaterale spontaneous (Ventil) pneumothorax durch drauerdrainage and pleuralyse. Mitt. d. Gesellsch. f. inn. Med. 35:144, 1936.

6. Vaccarezza, R. F., and Walinder, B. E.: A case of bilateral spontaneous pneumothorax caused by rupture of air vesicles in the lungs. Acta. Tuber. Scandinav. 10:66, 1936.

CEREBRAL PALSY TODAY*

LYON K. LOOMIS, M. D.

NEW ORLEANS

To realize where we are, it is sometimes interesting for us to see where we have been. In 1862, Dr. William John Little first described the spastic as the child with scissors gait who drooled and grimaced and was believed to be feebleminded. Because feeblemindedness was made a part of the description, the child was usually considered untreatable and placed in an institution to be forgotten. This attitude would probably still prevail if it were not for the untiring efforts of a few individuals who have greatly broadened our knowledge about cerebral palsy, among whom must certainly be mentioned Dr. Winthrop Phelps. We now realize that the spastic described by Little is only one of five types of cerebral palsy and we also know that feeblemindedness is often not present even in this type.

The present day interest in cerebral palsy is evident when reviewing the literature on the subject. During 1949, the number of articles in American medical literature approximately doubled the number in 1948. It has been said that cerebral palsy is rapidly becoming the tail that wags the dog in the broad field of work with crippled children.

TYPES OF CEREBRAL PALSY

The *spastic* type results from damage to the brain directly behind the frontal (thinking) area. Therefore, mental impairment is more common in this type than in any other type of cerebral palsy. When movements are attempted, the spastic muscle in opposition to the muscle being contracted, contracts itself instead of relaxing as it should. This is noted by the examiner as a stretch reflex and is characteristic of the true spastic.

The *athetoid* type is due to damage to the basal ganglia area of the brain, which is the filtering or sorting out area for voluntary motions. Thus, undesirable impulses

*Presented at Annual Convention of Louisiana Society for Crippled Children, November 1949.

are allowed to pass to the muscles controlling joints, bringing about involuntary motions. Mental impairment in this type of cerebral palsy is rare because the area of the brain affected is relatively more distant from the frontal area. Clinically athetosis is characterized by involuntary joint motions, and increase in nervous tension may exaggerate these movements.

Tremor is another type of cerebral palsy due to damage to the basal ganglia causing involuntary motions of reciprocal action with regular rhythm. Two types of tremor are noted clinically: (1) intention tremor in which the contractions occur only on attempted motion; and (2) nonintention tremor in which the contractions are present all of the time.

The *rigidity* type of cerebral palsy is caused by a diffuse brain lesion such as multiple petechial hemorrhages or encephalitis. Clinically these cases have stiffness in the extremities without involuntary motions or stretch reflexes. The affected muscles lack elasticity and a lead-pipe sensation is observed by the examiner when stretching is attempted by moving the joint.

The *ataxic* type of cerebral palsy is due to damage to the cerebellum which is the center of balance control. Mental impairment is rare in this type. Clinically, there is frequently a staggering sort of gait, the patient walking and standing with feet apart in order to provide a wider base of support. The affected muscles are hypotonic and reflexes may be diminished. Because the voluntary cerebral motor centers are able to take over the function of the damaged cerebellum, ataxics are very amenable to training.

OBJECTS OF TREATMENT

(1) *To make the individual useful to society.* (2) *To enable the individual to help himself.* (3) *To make the individual happier.* If these objects are always kept in mind, it will readily be seen that most cases of cerebral palsy require treatment. Even if the case is extremely severe and homebound so that we know it is impossible for him to be educated to hold a job, we are still obligated to train the patient to do little things for himself, which will make it easier for those who must care for him constantly. Even if these cannot be taught, we are still obligated to educate the patient in order to make him a happier individual providing he is educable.

SPEECH THERAPY

In treating the case, it must be remembered that the individual with cerebral palsy has five extremities: *one speech, two arms and two legs.*[3] The most important extremity is speech. Speech defects are frequently made the object of ridicule, although a crippling defect in the arm and leg is seldom held ridiculous. Since speech defects occur in 60 to 80 per cent of cases of cerebral palsy, we can readily see the need for proper emphasis upon speech therapy. We now know that speech defects are often brought about by incomplete shift of handedness. A moderate or mild degree of spasticity which can easily be overlooked or underestimated may exist in the dominant hand and if the child is made to use this hand with the thought that use will increase function, a severe frustration may be set up resulting in disturbed personality, speech, and learning. Dominance must be shifted completely to the good hand before rehabilitation is attempted.[5]

Good speech and use of the arms are certainly more important than spending all efforts to get the child to walk, yet how often we see the parent with the opposite viewpoint. It must always be remembered that many important positions can be held by persons with good speech and good arm function. Few positions can be held merely by the ability to walk.

PHYSIOTHERAPY AND OCCUPATIONAL THERAPY

The groundwork for rehabilitation of arms and legs is still occupational therapy and physical therapy, the former being most important for the arms and the latter most important for the legs. Various specific phases of these treatments have been called modalities by Phelps. There are fifteen different modalities for the five types of cerebral palsy:[3] massage, passive motion,

active assisted motion, active motion, resisted motion, conditioned motion, automatic or confused motion, combined motion, rest, relaxation, motion from the relaxed position, balance, reciprocation, reach and grasp, and skills. Some of these modalities are more important in one type of cerebral palsy than in another. For example, passive motion is more important in spastics, rigidities, and ataxics, but to less extent in athetoids and tremors. Relaxation is the basis of treatment in the athetoid but of no use in the ataxic. Motion from relaxed position is progression for the athetoid after he has mastered relaxation.

Some modalities are useful in all types of cerebral palsy among which may be listed the following: conditioned motion, combined motion, active motion, rest including supporting chairs, tables and braces, balance, reciprocation, reach and grasp, and skills.

SURGERY

Along with increased knowledge in the treatment of cerebral palsy by conservative methods has come greater caution in the use of surgery. Certain cases of the spastic type of cerebral palsy will need surgery after conservative treatment has failed. For example, a selective popliteal neurectomy is indicated on a patient of good mentality who is able to stand on the toes and has gastrocnemius and soleus spasticity resulting in knee flexion and equinus which cannot be corrected by physical therapy, night splints, or braces. Before doing this destructive operation, which will weaken chiefly the power to plantar flex, one must be certain that sufficient antagonist muscle power exists; otherwise, a flail foot will result and the patient may be worse off than before operation. The proper facilities for postoperative rehabilitation are imperative because proper splinting, bracing, and physical therapy after operation are often more important than the operation itself. Surgery is rarely necessary in athetosis and rigidities except to correct contractures. Surgery is practically never indicated in tremors and ataxias.

DRUGS

The efficacy of various drugs in the treatment of cerebral palsy is still a subject of much debate. At the present time it can definitely be said that for the most part drugs are only an adjunct to the more important methods of treatment. Prostigmine is still thought to have beneficial effect in the spastics and rigidities chiefly by aiding the finer motions such as speech and finger motions.[8] Dilantin appears to be helpful in athetosis and has advantage over barbiturates by relaxing without producing drowsiness. Myanesin (trade names sinan, oranixon and tolserol) has been reported helpful chiefly in athetoids,[1] but its efficacy has not been conclusively proved. In a recent report upon d-tubocurarine chloride (curare) in which 35 cases were treated, the authors felt that too much cannot be expected of the drug in the treatment of cerebral palsy.[4] The use of moccasin venom solution in a series of 100 cases was reported by Wheeldon who showed that there was subjective improvement in 50 per cent of the cases and objective improvement in 75 per cent[9] in athetosis, synkinesia and ataxia. Spasticity was not benefitted. Favorable results to date in the treatment of Parkinsonism with tri-hexyphenadil (artane) have been reported by Dow and Rosenbaum[2] in 8 out of 10 cases and it is their opinion that artane should be given a trial in every case of Parkinsonism. Schwab[6] and Tillman also report that artane alone and in combination with other drugs brought about alleviation of symptoms in 44 cases of Parkinson's disease. It is possible that artane will supplant the use of the hyoscyamine group which has been used considerably in the tremors. The ataxias should be given an abundance of B_6 and vitamin E which is probably an adjunct to improving muscle tone along with physical therapy.[7] Since children with cerebral palsy are frequently more nervous than normal, small doses of sedatives such as phenobarbital will frequently bring about slight general improvement as might be observed in any nervous patient. Improvement in general health by proper nutrition including vita-

mins must not be forgotten in the cerebral palsied as in the normal.

Slight improvement with the use of any new drug can frequently be expected from the psychological effect of trying something new. Remember that parents, patient and doctor are awaiting the miracle drug which will be the easy cure-all for cerebral palsy victims. To guard against overoptimism and the letdown which invariably follows when the new drug does not work the miracle, the doctor would do well to maintain a healthy skepticism toward all new drugs. This of course is difficult when laymen read of miracle cures in everyday print.

PSYCHOTHERAPY

The cerebral palsy patient is many times handicapped by his own personality, and if so, this must be influenced through training. Every cerebral palsy case is different from a psychological point of view and this must ever be kept in mind in treating the case by various methods. In general, the personalities of the five major types of cerebral palsy differ considerably. The spastic is usually an introvert whose life is dominated by fear, such as fear of loud noises, falling, and new surroundings. The athetoid on the other hand is usually an extrovert, has few fears, is quick to make friends and is aggressive. The ataxic resembles the athetoid in that fear is not highly developed and affection is highly developed. The intention tremor has a personality similar to the spastic and the nonintention tremor is more similar to the athetoid. Great consideration must be given to these various elements, especially fear, in the handling of the spastic case.

Occasionally consultation with the psychiatrist will be of great help in developing confidence to do certain things that seem almost unsurmountable. This may be true when the child is nearly able to stand by himself; proper psychotherapy may be of much aid in helping him pass the hurdle.

There is probably no other disease in which the proper relationship between parent and child is more important than in cerebral palsy. There can be no doubt but that the parents of the child with cerebral palsy should routinely take an intensive course to better understand their child and his mental and emotional makeup before attempting to embark upon the long voyage of living with him. If the wrong relationship is started early in life, the child may easily be spoiled from too much pity rather than neglected from too little love.

Recreation and hobbies are important. Summer camps are now being held in many parts of the country, which are geared to the life of the child with cerebral palsy. It gives the child something to look forward to and plan for as it does the normal child.

Photography can be as good a hobby for the cerebral palsied as it is for the normal for it gives opportunity for originality and expression. Of course, it may be necessary to improvise some special apparatus to hold the camera securely in position. Let us try to see that these children lead as well rounded and balanced a life as is possible.

REFERENCES

1. Cook W.: New concepts in management of cerebral palsy, J. South Carolina M. A., 45:248((Aug.) 1949.

2. Dow, R. A., Rosenbaum, H.: Treatment of Parkinsonism with artane, Northwest Med., 48:699, (Oct.) 1949.

3. Egel, P. F.: Technique of Treatment for the Cerebral Palsy Child, C. V. Mosby Co., 1948.

4. Hohm, C. L., Olsen, E. V., Dow, R. S.: Treatment of cerebral palsy. Northwest Med., 48:321, (May) 1949.

5. Phelps, W. M., Arthur, T. A.: Left hand, right hand, Hygeia, 26:808, 1948.

6. Schwab, R. S., Tillman, W. R.: Artane in treatment of Parkinson's Disease, New Eng. J. Med., 241:483; (Sept.) 1949.

7. Speed, J. S., Smith, H.: Campbell's Operative Orthopedics, Vol. II, 2d Ed., C. V. Mosby Co., 1949, pgs. 1476-1480.

8. Thomson, J. E. M.: Regional Orthopaedic Surgery and Fundamental Orthopaedic Problems, J. W. Edwards, 1947, pgs. 171-176.

9. Wheeldon, T. F.: Studies of Thomas F. Wheeldon on the use of venom in the treatment of spastic paralysis, manuscript.

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ORGANIZED MEDICINE AND MEDICAL EDUCATION

The interest and effort of the true physician has ever been to further the knowledge of the healing art, and to promote the well-being of his fellow man. One such purpose has been served for centuries past by teaching those who follow, and the other by instructing his patients. Out of this have grown medical schools, public health systems, and various agencies for study.

Over a half century ago, medical education was in a state of chaos. The title of "doctor" so impressed the public that patients did not look to discern how well qualified he was. Organized medicine undertook to improve and standardize medical teaching. The result was that the public was taught to applaud the acceptable medical schools, while diploma mills were legislated out of existence, or withered for want of students. About the same time, laws to control licenses to practice were encouraged and eventually established in all states. This effort on the part of the various units of organized medicine brought immeasurable good to society as a whole and is responsible for an appreciable part of the present improved state of health. This is evidenced, among other things, by an increased life expectancy of about sixteen years since the beginning of the century. In the meantime, medicine has become more valuable to the citizen and he wants more of it. Because it is expensive, the Communists are whispering into his ear that he can have all he wants if he will just let them arrange for his neighbor to pay for it.

At this juncture, organized medicine finds itself in a position of striving to educate the public on medical values and at the same time fight off legislation calculated to socialize medicine and bring on a medical dictatorship. To these ends an educational campaign of the AMA has been applied. The effectiveness has been gratifying to all concerned. The local units of the Society have supplemented the national campaign, and brought the average citizen to the realization of his dangers. Our campaign now, however, is threatened from an additional source. This is the grants-in-aid program now being carried out by the Federal Security Agency, to various medical schools. These grants are for the purpose of research. This research will, of course, mean support of staff and in turn will mean federal control of staff. In addition to this process, which is already going on, bills are being introduced, which would have the effect of direct grants from the federal Government to medical schools, and indirectly to the students themselves. These expenditures by a prodigal government can only mean complete control of medicine by the government. Students will grow up with the idea that federal support and bureaucratic medicine are the proper way of

life. The potentialities in this field stagger the imagination. It need only be recalled that when Hitler set out to accomplish his diabolical ends he wasted little time on the static forms of German society as it then existed, but took over the labor unions and started the indoctrination of the youth in the schools and colleges.

The AMA has taken a definite stand against such subsidy and control of medical schools by the government. For effectiveness of this stand a positive move will be necessary. To this end, it is suggested that the AMA subsidize the medical schools. Startling as this may be at first thought, it is thoroughly within keeping of the aims and potentialities of organized medicine. It is obvious that the expenses of maintaining a medical school have outrun the endowed resources and the students' pocketbooks. In order to live up to the standards that all wish to maintain, outside assistance could come from no more proper source than doctors themselves. We can better be depended upon to keep our own house in order, than we could to maintain a semblance of order when dominated by politicians.

The requested contribution or assessment of $25.00 from each member of the AMA brought in a huge fund of over $2,000,000 which has been used in the general educational campaign. This amount has been fixed as the dues for the members for the coming year. Medical dues in comparison to those of the monopolistic labor unions are strangely low. The average income in such unions will vary between $2000 and $5000 per year per individual. Recent figures have set the average income for the doctor at approximately $10,000 a year. To maintain the right of private practice, the average physician could well afford to pay much more of his income into a fund to be used in the general interest than the average laborer. Such a fund in the hands of organized medicine could very quickly grow to a point where it would dwarf the pools of national labor unions. Among its services to the doctors and to the public could be the subsidy of the medical schools. Who would know their needs any better than ourselves? The temptation to squander public tax money would be absent.

Under these conditions, the schools would be receptive to principles of private practice, which in some areas is not the case now. Students would then look to organized medicine rather than to a prodigal government as the source of the funds which provided their education.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

DO NOT LAY DOWN YOUR ARMS

Recent developments in Washington, especially in the halls of Congress, and from the executive offices of the President indicate that the administration is preparing to put on one of the greatest fights in the history of this country to carry out their determination to federalize medicine. They have tried in a disguised manner to give the medical profession and the public sufficient opiate to desensitize them, by various rumors and expressions from leading senators *that there will be no action on federalization of medicine in 1950.* Do not let us be led into such a complacent disposition. Mr. Oscar R. Ewing, Director of Social Security, is not going to Europe just for his health. He is going there for the express purpose of seeing if it is possible to find some advantageous developments which would bolster up the cause of federalized medicine. Accompanying him on this trip are many of his satellites and co-workers who will spread all over Europe among the socialistic states to find out how well

their compulsory medical plan is working. You may expect these individuals to bring back only those results which have been most appreciated by the public, and not the other side where medicine has been brought to the lowest ebb since civilization.

There are several bills now in a formidable position in the present Congress which, if acted upon favorably, will go a long way toward the enactment of federalized medicine. I particularly refer to Senate Bill No. 1411 known as the School Health Bill, in which it is proposed that the government examine and treat some 29,000,000 school children free of charge, without any consideration as to individual economic status. Senate Bill No. 1453 is of a similar nature, but provides benefits which give subsidy to medical schools after meeting certain qualifications for construction and enlargement of staff facilities, and this bill also provides a royalty to every student studying medicine. There is no test as to the economic status of the institution or of the individual medical student. Do we wish to have our public schools controlled and regulated by the federal government? Do we wish to have our medical schools and doctors subsidized? Do you wish them to be the pawns of the federal government? All of this would merely reduce to a minimum the independence of our institutions, as well as of the individual doctor.

We are sending the above message to you more or less in the spirit of warning you not to let up one iota in the wonderful work you have done in this state toward preventing passage of any compulsory health insurance. Probably the next two or three years will be much more trying and results more decisive than in any previous twenty years of medical history. We must stay united and fight with all the power we have; with honesty, integrity and assurance of our cause being the American way. This is a fight for the public to understand as the consequence of such tyrannical legislation would be most destructive to their freedoms and liberties and destroy free enterprise for all which we have been so proud of in the past. The successful passage of federalized medicine is but a spoke in the wheel of the welfare state, which we must stop now or be in the tentacles of the octupus of the socialistic state. Attendance at national medical meetings during the past four or five months is very convincing that the medical profession is very conscious of the situation and is developing plans for a continuous fight to forestall the realization of victory by our opponents.

In the words of Mr. Oscar R. Ewing: "Our campaign plan is to educate each of the 3,000 Democratic precinct committeemen and women in all details of the National Health Insurance plan. They will explain it to the people." However, Dr. George F. Lull, secretary and general manager of the American Medical Association, states the following: "We will have to carry on this fight until someone is licked. It is false to ourselves and false to the medical profession to take the attitude that there is no need to continue an active campaign." So let's keep up the fight until the victory is won.

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REPORT ON PROGRESS OF LOUISIANA PHYSICIANS SERVICE

It has just been three years since the committee on voluntary health insurance of the Louisiana State Medical Society organized Louisiana Physicians Service, Inc., the Blue Shield Plan. Through the establishment of this organization the doctors have been able to make available to the public a system of voluntary medical, surgical and obstetrical care.

The first contracts were issued November 1, 1946. Looking at their report at the end of operations on October 31, 1949, or just three years after the company was organized, we find that nearly 40,000 people are now covered and eligible to receive Blue Shield benefits. The income has increased considerably each year to where now the company will collect next year, more than $300,000.00 in premiums, of which 70 to 80% of this amount will be distributed to the doctors of this state for services rendered the subscribers. During the three

year period 9,062 claims were presented for services performed by the doctors of this state. These claims amounted to nearly $400,000.00.

The month of October 1949 was the largest single month for enrollment increase by the company. During October 5,382 persons were enrolled, 2,804 of these being through group enrollment and 2,578 persons being enrolled through the individual enrollment campaign conducted in late September and early October.

The first concentrated advertising campaign conducted by Blue Cross and Blue Shield was during the individual enrollment campaign. Newspapers throughout the state carried Blue Cross, Blue Shield ads for more than two weeks and radio stations throughout the entire state carried public announcements, urging people to enroll in Blue Cross, Blue Shield. Such advertising speaks for itself in increased enrollment as evidenced by the figures previously quoted.

The Council on Medical Service and Public Relations of the Louisiana State Medical Society participated actively in the advertising and promotion program for Blue Cross and Blue Shield.

The secretary of your society felt that this was an opportune time to present a brief report on Louisiana Physicians Service. A more detailed report will be rendered at the close of 1949. The Board of Directors of Louisiana Physicians Service has asked the Journal to express its sincerest thanks to all doctors throughout Louisiana for their wholehearted participation in the State Society's voluntary health insurance program.

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LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

ANNUAL MEETING
SURGICAL ASSOCIATION OF LOUISIANA

The Surgical Association of Louisiana held its second annual meeting, at the St. Charles Hotel, on Friday, November 11, 1949. There were three papers in the morning and two round table discussions in the afternoon:

Relationship between Sex Hormones and Breast Carcinoma by Dr. Paul D. Abramson, Shreveport.

Certain Essential Considerations in the Management of Postoperative Thrombosis and Embolism by Dr. William H. Parsons, Vicksburg, Mississippi.

Annual address—Carcinoma of the Cervix by Dr. J. H. Pratt, Rochester, Minnesota.

Symposium—Acute Abdominal Emergencies.

Moderator—Dr. J. H. Pratt.

Associates—Dr. Lawrence H. Strug, Dr. Geo. Wm. Wright.

Symposium—Malignant Tumors.

Moderator—Dr. Ambrose H. Storck.

Associates—Dr. Wm. H. Harris, Jr., Dr. Samuel Romano, Dr. L. Sydney Charbonnet, Jr.

Dr. Isidore Cohn, New Orleans, was elected president for the coming year. Other officers elected are as follows: Dr. James Q. Graves, Monroe, first vice president, and Dr. Arthur N. Houston, New Orleans, second vice president; Dr. Henry G. Butker, New Orleans, secretary; Dr. Edmund L. Leckert, New Orleans, treasurer.

The members elected this year are: Dr. Charles L. Black, Shreveport; Dr. Henson S. Coon, Monroe; Dr. Claude C. Craighead, New Orleans; Dr. James H. Eddy, Jr., Shreveport; Dr. Marvin T. Green, Ruston; Dr. James E. McConnell, Monroe; Dr. Walter W. McCook, Jr., Shreveport; Dr. Richard E. C. Miller, Alexandria; Dr. Hollis T. Rogers, Winnsboro; Dr. Leonard H. Stander, Baton Rouge; Dr. Harry M. Trifon, Shreveport; Dr. Myron A. Walker, Baton Rouge; Dr. James S. Webb, Jr., New Orleans.

The growth and strength of the Surgical Association of Louisiana in the past year has been most creditable. It is serving a valuable purpose in promoting the standards of surgery and hospital care in our State. Its continued success is heartening.

ANNUAL MEETING OF THE LOUISIANA ACADEMY OF GENERAL PRACTICE

The Louisiana Academy of General Practice held its annual meeting in Alexandria, on December 11 and 12, at the Hotel Bentley, Dr. D. B. Barber, president, presiding. The General Chairman of the meeting was Dr. C. P. Herrington of Alexandria. Doctors Esmond Fatter, New Orleans, Joel P. Gray, New Orleans; J. P. Sanders, Shreveport; John W. Atkinson, Gretna, were in charge of the commercial exhibits.

After registration there was a business meeting of the delegates, followed by dinner and an address of welcome by the president of the Eighth District, Dr. James Knoll, Bunkie.

On Monday, December 12, the following scientific program, arranged by the Chairman of the Program Committee, Dr. J. A. White, Jr., was presented.

Schizophrenia, by Dr. Howard G. Alexander, Staff, Central Louisiana Hospital, Pineville.

Mediastinal Tumors by Dr. E. C. Uhrich, Pathologist, Baptist Hospital, Shreveport.

Chronic, Nonspecific Ulcerative Colitis by Dr. Charles A. Jones, Chief, Department of Medicine, U. S. Veterans Hospital, New Orleans.

Pelvic Tumors that May Obstruct the Birth Canal by Dr. Sim B. Lovelady (recently Dept. Gyn., Mayo Clinic), Houston, Texas.

Ear, Nose, and Throat Allergy in General Practice by Dr. Jack R. Anderson, Staff of Eye, Ear, Nose & Throat Hospital, New Orleans.

Leprosy (with Kodachrome slides) by Dr. Rolla R. Wolcott, Senior Surgeon, U. S. Marine Hospital, Carville, La.

Socialized Medicine in England by Dr. Hugh Smith, Associate Professor of Orthopedics, University of Tennessee. (Dr. Smith spent six weeks in England recently on a commission making a study of this subject).

Diagnosis and Management of Occiput Posterior Position by Dr. E. L. King, Professor of Obstetrics, Tulane University, New Orleans.

WOMAN'S AUXILIARY

After having been inactive over the summer months, the various local auxiliaries have begun their fall meetings. The Woman's Auxiliary to the Orleans Parish Medical Society met in October at the Orleans Club. Mrs. Herman Gessner, Medical Cultural Items Chairman, was the program chairman of the afternoon. She introduced Miss Mary Louise Marshall, librarian of the Rudolph Matas Medical Library of Tulane University, and assistant librarian of the Orleans Parish Medical Society. Miss Marshall spoke on "The Romance of Medicine in New Orleans."

The Woman's Auxiliary to the East Baton Rouge Parish met at the Woman's Clubhouse in October. The local points of interest were the committee reports and plans for next year's state convention. Also on the agenda was the showing of the Community Chest film, "This is Our Town."

The program of the first meetings of the Woman's Auxiliary to Rapides Parish Medical Society was devoted to a discussion of our publications. Mrs. O. B. Owens, editor of News and Views and immediate past president of the State Auxiliary, discussed both the National Bulletin and News and Views. She read excerpts from the bulletin, particularly in regard to the educational campaign being conducted by Whitaker and Baxter, and to the 12 point program of AMA.

Chairmen of publicity in the local auxiliaries are reminded to send notices of meetings to the State Chairman of Press and Publicity. Of interest, also, is information about special projects such as a Speakers Bureau and essay contests.

Mrs. Daniel M. Kingsley, Chairman,
Press and Publicity.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

WOMAN'S AUXILIARY TO THE LOUISIANA ACADEMY OF GENERAL PRACTICE

December 11 and 12 are red letter dates for the wives of General Practitioners in Louisiana. With their husbands who will meet in Alexandria at that time, they are going to be very occupied in making new acquaintanceships and renewing old friendships.

Except for the board members who will convene for a short session Sunday afternoon, the auxiliary is inaugurating a procedure of assembly without business meetings. Just come, attend a few parties, talk with your friends, and relax from the many, many duties a doctor's wife is heir to.

Due to the fact that the convention date of the Louisiana Academy of General Practice has been changed to the fall so as not to interfere with attendance at the Louisiana State Medical Society meetings, traditionally held in the spring, there will be no necessity for business sessions for the entire group. The board of the L.A.G.P. has decided that the incumbent officers will serve the long term until the fall of 1950, so the women's group will of course follow the same procedure.

Enterprising Mrs. D. B. Barber, the president of the group, will enjoy the fortunate circumstance

of being able to relax as she presides at an assembly of auxiliary members in her own home town.

With the aid of her Louisiana state board members, Mrs. Barber corresponded with every state president where an Academy of General Practice was organized, with their wives, with every National Officer of the American Academy of General Practice, every board member, their wives also, gathering opinions as to the advisability of organizing a National Auxiliary at the meeting scheduled for February 21, 22 and 23 in St. Louis. The National Board discussed this question at the Mid-Year Board meeting, which was held in Kansas City in October, and decided that the Academy of General Practice was yet too young to sponsor an auxiliary.

Nevertheless the women of the Louisiana Auxiliary, the first in the nation to organize, feel that in the very near future, every state will have an organized auxiliary, which will of necessity initiate proceedings for national organization.

The aim of the auxiliary as presently organized is twofold; to promote the general practice program and to assist the doctor husbands with their projects, and secondly, to promote better acquaintanceship among the wives and families of the general practitioner.

The very informal program as planned for the Alexandria conclave starts with a leisurely drive or train trip to Alexandria on Sunday. There will be only a board meeting for the women on Sunday afternoon, but the Presidents Barber (heads of the men's and women's group respectively) will entertain the member-friends of the dual organization at a cocktail party in the Bentley Hotel Sunday evening, to be followed by the traditional doctor-wife get together.

Monday the auxiliary members will lunch together with the afternoon free for shopping, chatting, resting or sight-seeing. Monday night in formal attire (for the ladies only) the final dinner meeting of the group will be held. This dress affair (which every woman loves) will be the only note of formality to the whole affair. Just come with your husband, whether or not you are a registered member of the state group. We'll see that you are asking to join before Monday noon.

Place—Alexandria.

Dates—December 11 and 12.

Who—Every general practitioner's wife in Louisiana.

Why—To be with your husband and to meet all the other G-P wives.

What—Just get together and fun. No business meetings.

Mazie Adkins Guidry,
Publicity Chairman.

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BOOK REVIEWS

Outwitting Your Years: By Clarance William Lieb, M. A., M. D., New York, Prentice-Hall, Inc., 1949, pp. 278. Price, $2.75.

This is one of the many books purporting to act as guide to a healthier, happier life for the ageing and aged individual. The author has combined a thorough knowledge of the subject of geriatrics with good common sense and presented it in a witty, pithy fashion, readily understood by the layman.

I. L. Robbins, M. D.

Marihuana in Latin America: The Threat It Constitutes: By Pablo Osvaldo Wolff, M. D., Ph. D., M. A., Washington, D. C., The Linacre Press, Inc., 1949, pp. 56. Price, $1.50.

The author of this monograph is a member of the Expert Committee on Habit Forming Drugs of the World Health Organization and apparently is in close touch with the problems of drug addiction in which he has been interested for a considerable period of time. Particular attention, in his discussion, is paid to the sociologic and especially criminal aspects of the marihuana habit. Most of the information concerns the conditions existing in Brazil, Mexico, and Cuba, although references are also made to the use of the narcotic in certain other Latin-American countries and in the United States. On the whole, much more importance is attributed to the use of marihuana in the instigation of crime and in the promotion of mental and physical deterioration than has been given in certain other publications, notably that of the La-Guardia Committee based on a general survey and experimental study in New York City. There can be little doubt that the acute effects of the drug may lead in certain instances to the commission of crimes which would not otherwise occur and that the effects may be used by criminals as an abetting agent in carrying out premeditated crimes. The marihuana habit is also commonly encountered in habitual criminals. The author is also convinced that its chronic use leads to mental, moral, and physical deterioration involving the creation of criminal attitudes and acts.

In the opinion of the reviewer, conclusions regarding the relative importance of marihuana in the causation of crime encounter the same difficulties as those experienced with the use of alcohol. In other words, sufficiently reliable and extensive data must be obtained and then the data must be evaluated in its relation to the occurrence of crime as a whole. There is also the difficulty of determining the part played by other stigmata and extrane-

ous factors, such as mental defects, economic level and, environment, occurring concurrently in individual cases. Is the drug habit a primary cause of crime and criminal tendencies or is it but another manifestation of more deep-seated causes? These aspects are minimized by the author.

In addition to the above phase of the subject, useful and interesting information is also given on the wide extent of the habit, the general effects of the drug, and custom associated with its use. Distinction is made between this habit and true addiction produced by the opiates in that only slight tolerance is developed to marihuana and that dependence is mainly of a psychic and emotional type rather than physical. Throughout the monograph a very strong stand is taken on the dangers involved in the marihuana habit and the importance of eradicating it. An extensive bibliography is included.

RALPH G. SMITH, M. D.

Interns' Manual: By American Medical Association. Philadelphia, W. B. Saunders Co., 1948, pp. 201. Price, $2.25.

This compact little volume, successor to "Hospital Practice for Interns," has been prepared by various Councils and Bureaus of the American Medical Association, and compiled and edited in the office of the Council on Pharmacy and Chemistry. It contains much useful information essential to the young intern, such as his place in the hospital, the lawful scope of his practice in each state, and the services of the American Medical Association available to him.

The clinical data comprise emergencies such as shock, diabetic coma, gastric hemorrhage, treatment of hemorrhage after tonsillectomy, etc. One chapter is devoted to acute poisoning, with description of symptoms and treatment of more than 50 types of poisoning. The latest uses and dosages of 248 proven drugs are given, with prescriptions, tables of solubility, and metric-apothecary equivalents. Standard tests are included, and normal values are given for blood, urine, spinal fluid, and gastric contents. Many diet tables are included.

I. L. ROBBINS, M. D.

Cardiovascular Disease: By Louis H. Sigler, M. D., F. A. C. P., New York, Grune & Stratton, 1949, pp. 551, illus. Price, $10.00.

This book offers an excellent review of the significant factor concerning various features of cardiovascular disease. The author has successfully achieved his objective of covering the field of cardiovascular disease with emphasis on the pathologic physiology of disease as elucidated in the investigations of the past few years and the treatment of the patient as an individual with particular reference to psychosomatic aspects of his disease. Of particular interest are the first three chapters concerning the general incidence and types of cardiovascular disease as well as a fairly detailed presentation of the anatomy and histology of the heart and blood vessels. These data are not usually found in textbooks of cardiology.

The rather frequent generalizations, which are at times inaccurate, are a poor feature of the book. Nevertheless, the book is recommended to anyone who desires an easily read review of cardiovascular disease written from the clinical standpoint.

FRANK J. KELLY.

American Nurses' Dictionary; the definition and pronunciation of terms in the nursing vocabulary: By Alice L. Price, B.S., R.N. Philadelphia, W. B. Saunders Co., 1949., pp. 655 plus 101. Price, $3.75.

Included in this medical dictionary are approximately 25,000 words chosen primarily to meet the requirements of the nurse. Pronunciation and definition are given clearly and concisely. No etymology is included. Appendices contain lists of abbreviations commonly used in medicine, prefixes, suffixes, symbols; tables of the arteries, bones, chemical elements, muscles, cranial nerves and the veins.

Supplementing the Dictionary is a Teacher's Vocabulary guide in pamphlet form in which are grouped by subject the most important words which the nurse should know.

This volume will fill a very useful purpose.

MARY LOUISE MARSHALL.

Handbook of Orthopaedic Surgery: By Alfred Rives Shands, Jr., B.A., M.D. 3rd ed. St. Louis, The C. V. Mosby Company, 1948, pp. 574. Illus. Price, $6.00.

The author states in the preface of this third edition that his book has become more or less the standard text for teaching of graduate and undergraduate orthopedic teaching, as well as being used extensively by the general practitioner as a reference book in orthopedic surgery. The third edition represents considerable expansion over the second edition with the re-insertion of numerous illustrations which were deleted from the second edition, having appeared only in the first edition of the book. The illustrations are of good quality, although the use of drawings to depict x-ray changes is no more successful here than they are elsewhere in medical literature. The material in the text is presented concisely and in a definite outline form which lends itself very well to its use by undergraduate students. One of the most valuable features of the instruction to students is the illustration of improper methods of immobilization or positioning of patients, as compared to the proper method of immobilizing patients, with an

excellent discussion of the reason for the positions of immobilization, as illustrated.

The incorporation of extensive material from current literature and the compilation of an accurate and extensive bibliography increase the value of this text to the students immeasurably.

JACK WICKSTROM, M.D.

Physician's Handbook: By John Warkentin, Ph.D., M.D., and Jack D. Lange, M.S., M.D. 5th ed. Palo Alto, Calif., University Medical Publishers, 1948. Pp. 294, illus. Price, $2.00.

This handbook summarizes diagnostic procedures and factual data which a physician must have quickly available. The scope of the 5th edition has been extended in order to be a serviceable pocket-reference for many types of medical practice. Most of the standard laboratory and clinical tests are included.

In this revision the authors have given emph to clinical factors, such as the significance of laboratory test findings. This is evidenced in the sections on the comatose patient, urine, blood and liver tests, the hormones, and diagnosis of poisoning. An autopsy outline and treatment of acute poisoning and alcoholic intoxication have been added. Simplifications of older tests are also included, such as the "newspaper test" for urine sulfonamides.

I. L. ROBBINS, M.D.

Clinical Auscultation of the Heart: By Samuel A. Levine, M.D., and W. Proctor Harvey, M.D. Philadelphia, W. B. Saunders Co., 1949. Pp. 327. Illus. Price, $6.50.

The authors state in the preface of this treatise that they have been impressed by the fact that many physicians, not only general practitioners but also able internists and even cardiologists, were not applying all the information that can be obtained by simple auscultation. For this reason they believed that this detailed discussion of bedside auscultation would be timely. The reviewer is sure that those physicians who read the book will agree.

The authors discuss normal heart sounds and deductions which may be made from changes in character of these sounds. They discuss the recognition of the various cardiac arrhythmias by bedside auscultation, the interpretations possible from different murmurs and numerous miscellaneous ausculatory findings such as venous hums, Duroziez's signs, etc. The various acoustic phenomena are discussed along with the clinical significance of the particular finding. Illustrations by actual sound tracings greatly clarify the presentation and add much to the book.

Any physician who carries a stethoscope will find reading this concise, well illustrated monograph a valuable experience.

CHARLES E. JACKSON, M. D.

PUBLICATIONS RECEIVED

Charles C. Thomas, Springfield, Illinois: The Diagnosis and Treatment of Adrenal Insufficiency by George W. Thorn, M.D., with the collaboration of Peter H. Forsham, M.D., and Kendall Emerson, Jr., M.D. Resuscitation and Anesthesia for Wounded Men, by Henry K. Beecher, A.M., M.D.

W. B. Saunders, Philadelphia: Normal Values in Clinical Medicine, by F. William Sunderman, M.D. and Frederick Boerner, V.M.D. Diseases of the Heart, by Charles K. Friedberg, M.D. The Eye and Its Diseases, by Ninety-Two International Authorities, Edited by Conrad Berens, M.D., F.A.C.S., 2nd Edition. An Atlas of the Blood and Bone Marrow, by R. Philip Custer, M.D.

John C. Winston Company, Philadelphia: For the New Mother, by Mildred V. Hardcastle, R.N.

Froben Press, Inc., New York: Your Nasal Sinuses and Their Disorders, by Albert P. Seltzer, M.D.